



# ELECTROCARDIOGRAPHIC TEST BOOK

## VOLUME II

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# ELECTROCARDIOGRAPHIC TEST BOOK

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## PREFACE

Volume II is divided into Parts A and B. Answers to the questions and discussion of the tracings presented in Volume I, Part A, can be found on the corresponding pages in this volume. Answers to Questions on Electrocardiographic Interpretation can be found in Part B of this volume.

For purposes of clarity the answers and explanations are brief and do not represent a complete discussion of each subject. For a more detailed discussion the reader should refer to the standard text books on electrocardiography.

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PART A

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EXPLANATIONS OF ELECTROCARDIOGRAMS

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## Answers

I E

II A

## Explanation

The electrocardiographic position of the heart is horizontal. The ventricular complexes of lead  $aV_L$  resemble those of leads  $V_5$  and  $V_6$ . The ventricular complexes of lead  $aV_F$  resemble those of leads  $V_1$  and  $V_2$ .

The electric axis of the QRS complexes is minus 30 degrees (limit of normal is minus 30 degrees; see appendix). Often heart disease is present when the electric axis is more negative than minus 30 degrees; however, a few normal individuals have an axis more negative than this. There is no definite electrocardiographic evidence of cardiac disease.

## Case Summary

Arteriosclerosis of the thoracic aorta is present. Average blood pressure 140/90. Advanced arteriosclerosis of the retinal arteries. The heart is enlarged to the left. The cardiothoracic ratio is 0.50.

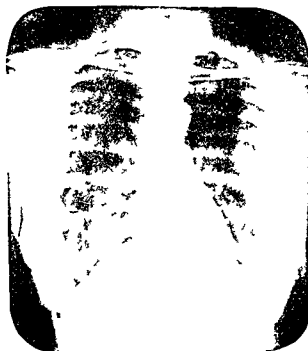


ation

normal sinus rhythm is present as the cardiac rate is between 60 and 100 beats per minute  
ing 63 beats per minute and the pacemaker is in the sinus node

ummary

aroxysmal essential hypertension Average blood pressure 160/80 The heart is not enlarged  
he aortic knob is prominent The cardiothoracic ratio is 0.47 There is a Ghon tubercle in  
ie right outer lower lung field



C

*Explanation*

The negative S T segments in leads  $V_5$  and  $V_6$  and other leads are consistent with subendocardial injury. This may be secondary to coronary artery disease. The Q T intervals are not shortened when the segment shifts are due to subendocardial injury but they are shortened when due to digitalis. The inverted T waves in lead  $V_4$  suggest myocardial ischemia. Certain features of this tracing resemble left ventricular hypertrophy; however, there is not sufficient evidence of left ventricular hypertrophy to make such a diagnosis with certainty.

*Case Summary*

Coronary arteriosclerosis with coronary insufficiency and angina pectoris. The highest blood pressure was 158/80. Teleoroentgenogram shows the heart is normal size, shape, and position and the cardiothoracic ratio is 0.38. At no time has this patient had digitalis or digitalis-like drugs nor has there been an electrolyte disturbance. Thus the abnormal sagging of the S T segments in leads  $V_5$  and  $V_6$  is in keeping with typical angina of effort and is due probably to myocardial ischemia and injury.

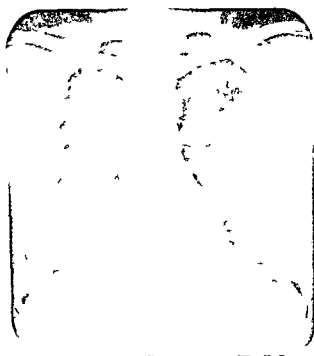


ation

o significant electrocardiographic abnormalities Slight notching of the P waves occur in significant number of normal subjects The S T segments in lead  $aV_F$  do not sag sufficiently to be outside of normal range

### Summary

Normal male No heart disease Heart normal size shape and position The cardiothoracic ratio is 0.39



Answer

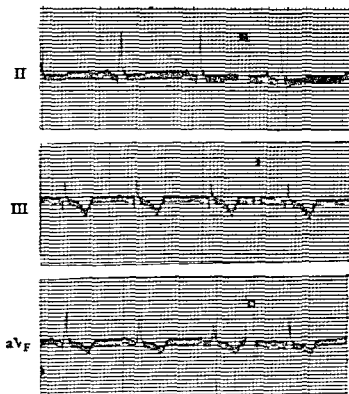
B

Explanation

Injury to the posterior wall of the left ventricle is suggested by the low T waves in lead II (lower limit of normal 1.0 mm.) and abnormal T waves in lead III (lower limit of normal minus 1 mm. see appendix). Sagging of the S-T segments is present in lead aV<sub>F</sub>. A tracing taken thirty days previously shows isoelectric T waves in lead II and deeply inverted T waves in leads III and aV<sub>F</sub>. (See below)

Case Summary

Coronary arteriosclerosis. Possible myocardial infarct four months previously associated with increased sedimentation rate and white count. Teleoroentgenogram shows the heart to be at the upper limit of normal in size. The cardiothoracic ratio is 0.46



Tracing taken 20 days previously  
which was 3 months after a  
possible myocardial infarct

## Answers

I C

II C

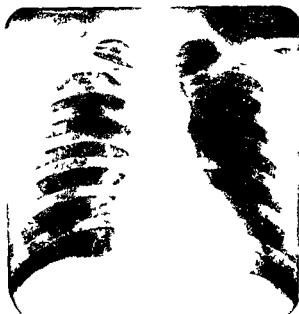
## Explanation

The electrocardiographic position is intermediate. The ventricular complexes of leads  $aV_L$  and  $aV_F$  are similar in form and size and like those of leads  $V_5$  and  $V_6$ .

The tracing is abnormal because of the QRS complexes of increased width which measure 0.12 mm in lead II. This could be secondary to ventricular hypertrophy or could represent the early development of an intraventricular block.

## Case Summary

Periarteritis nodosa diagnosed from biopsy with arterial hypertension. Heart upper limit of normal size. Cardiothoracic ratio is 0.47. The average blood pressure 148/110.



## Answers

I B

II B

## Explanation

The cardiac rate is 136 beats per minute and the cardiac pacemaker apparently is in the sinus node. In children of this age the heart rate should not exceed 109 beats per minute (see appendix).

Characteristically ST segment shifts occur when the cardiac rate is rapid. This occurs often because the P waves and atrial T waves are superimposed on the downstroke of the T waves producing apparent negative ST segment shifts. In this tracing, the P waves in the standard leads are peaked and are consistent with atrial enlargement. Also the notching of the S waves in lead  $V_1$  is consistent with but not diagnostic of an incomplete right bundle branch block.

## Case Summary

Minimal pulmonary stenosis with poststenotic dilation of the pulmonary artery. There was a grade III pulmonary systolic murmur and thrill. The teleroentgenogram shows fullness in the region of the pulmonary arteries with relatively clear peripheral lung fields. Cardiac catheterization revealed a slight increase in right ventricular pressure and a decrease in pulmonary artery pressure. This tracing is typical of a minimal amount of pulmonary stenosis. Larger amounts of stenosis produce electrocardiographic evidence of a right bundle branch block or of right ventricular hypertrophy.





## Answers

I B

II C

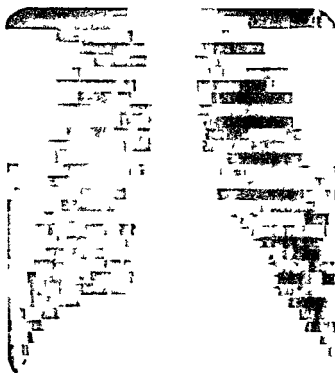
## Explanation

Sinus bradycardia is present because the cardiac rate is below 60 beats per minute and the atrial impulses originate in the sinus node. This is characteristic of the well trained athlete but is seen in a large number of normal subjects and in patients with hypothyroidism, vagotonia or cerebral arteriosclerosis.

The electrocardiographic position of the heart is vertical because the ventricular complexes of lead  $aV_L$  are similar to those of lead  $V_1$  and the ventricular complexes of lead  $aV_F$  are similar to those of  $V_6$ . This is a typical heart position of marathon runners.

## Case Summary

No heart disease. The patient is a well trained endurance athlete. X ray kymogram of the chest shows that the heart is at the upper limit of normal in size.



## Answers

I A

II B

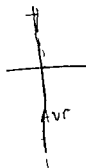
## Explanation

### Normal tracing

The electrocardiographic position of the heart is semihorizontal because the ventricular complexes of lead  $aV_L$  resemble those of leads  $V_5$  and  $V_6$  and the ventricular complexes of lead  $aV_F$  are small

### Case Summary

Intercostal neuritis No cardiac disease Heart normal size shape and position The cardio thoracic ratio is 0.43



Answer

C

*Explanation*

Small Q waves such as those in lead aV<sub>F</sub> are characteristic of an intermediate semivertical or vertically placed heart and represent normal ventricular depolarization. To be diagnostic of posterior infarction the Q waves in lead aV<sub>F</sub> should be 0.04 second in duration or greater and should exceed 28 per cent of the height of the R waves in this lead (see appendix). Sinus bradycardia rate 59 beats per minute.

*Case Summary*

Hyperlipemic xanthomatosis. Blood cholesterol 450 mg per cent. No heart disease. Heart normal size, shape and position. The cardiothoracic ratio is 0.42.



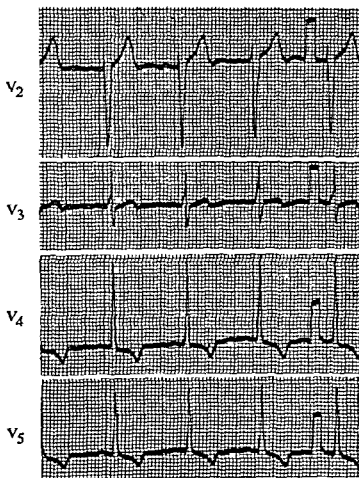
B

*Explanation*

High voltage of the R waves in lead I (greater than 11 mm) and discordant J points, S T segments and T waves suggest left ventricular hypertrophy. The Q waves in lead  $V_6$  exclude the possibility of a complete left bundle branch block. The negative S T segment shifts suggest subendocardial injury in addition to the left ventricular hypertrophy although typically left ventricular hypertrophy is associated with S T segments and T waves which are discordant with the major deflections of the QRS complexes. Subendocardial injury is supported by comparison with a previous tracing recorded one month before the attack of pulmonary edema (see below).

*Case Summary*

Hypertensive heart disease for ten years. Average blood pressure 190/110. One attack of pulmonary edema six months ago. Chest pain at that time. The teleoroentgenogram shows rounding of the apex of the heart. The cardiothoracic ratio is 0.47. There is widening of the great vessel shadow.



5 months previously



I C

II A

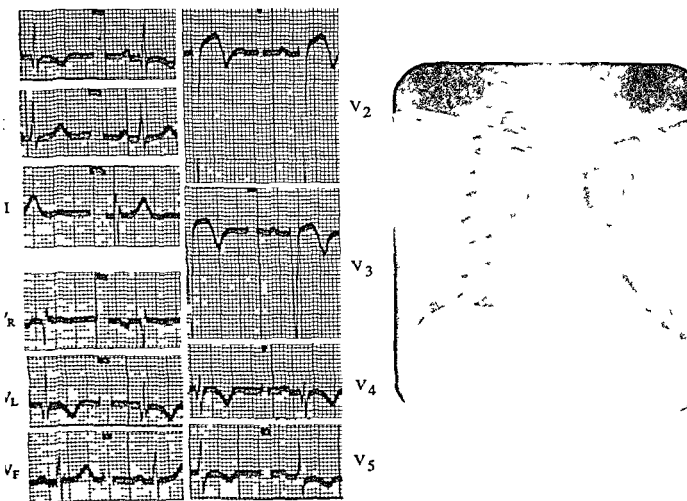
*Explanation*

The tremor is present in the left arm and is revealed by high frequency waves on the base line of lead  $aV_L$ . These waves appear in leads I and III as the left arm is common to each of these leads.

An old anterior infarct is suggested by the low R waves in lead  $V_4$ . They measure 1 mm (lower limit of normal 4 mm see appendix). This diagnosis is supported by the tracing recorded three years previously during the active stage of the anterior infarct (see below). The tracing about which the questions were asked shows in addition inverted T waves in leads II, III and  $aV_F$ , high T waves in leads  $V_1$ ,  $V_2$  and  $V_3$ , low T waves in lead  $V_6$ , large Q waves in lead III and low R waves in lead  $aV_F$  which suggest at least posterior myocardial injury and ischemia.

*Case Summary*

Anterior infarct three years ago. Posterior infarct one year ago. Heart normal size, shape and position. The great vessel shadow is widened slightly. The cardiothoracic ratio is 0.47.



Tracing taken 3 years ago

I D

II B

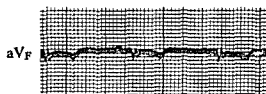
### Explanation

Muscle tremor is present in the unipolar lead  $aV_F$  and in the bipolar leads II and III. The tremor is in the left leg which is common to leads II and III. The tremor would not be expected in lead I which makes connections between the right and left arms. There is no tremor in leads  $aV_R$  or  $aV_L$ .

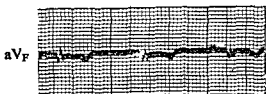
The Q waves and inverted T waves in lead  $aV_F$  and the flat T waves in lead II are consistent with an old posterior myocardial infarct.

### Case Summary

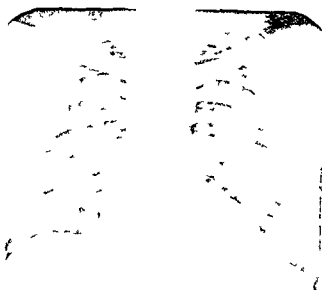
The patient had a myocardial infarct ten years ago for which he was hospitalized. Lead  $aV_F$  with normal breathing and after inspiration is shown below. These confirm the diagnosis of a posterior infarct because the Q waves remain essentially unchanged during deep inspiration. The teleroentgenogram shows that the heart is enlarged in its transverse diameter and there is rounding of the heart at the apex indicating left ventricular enlargement which was not revealed in the electrocardiogram.



Normal Breathing



Inspiration



Tracing taken three months previously

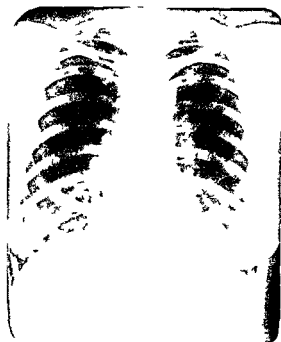
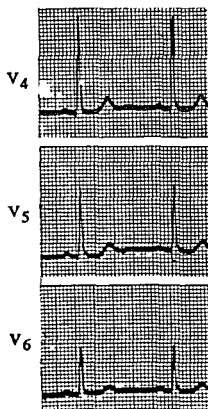
C

*Explanation*

Sagging of the ST segments is present in leads  $V_4$  through  $V_6$ . The segments sag about 0.2 mm (20 microvolts) below the abscissa which passes through the J points (junctions of the ending of the QRS complexes with the beginning of the ST segments). This is an uncommon finding among normal individuals. Sagging is the result often of a secondary change associated with left ventricular hypertrophy or it may be a primary change resulting from a subendocardial infarct, subendocardial injury, drugs, electrolyte disturbances or other causes. When the abnormality is due to left ventricular hypertrophy, the ST segment shifts persist often for years. With a subendocardial infarct, they persist for weeks or months and with acute subendocardial injury as occurs with angina pectoris, they last for minutes. In this patient, a second electrocardiogram taken one month later showed that the ST segment shifts had returned to normal (see below). The tracing is not suggestive of pericarditis. The time of left ventricular depolarization is within normal limits as the time of occurrence of the intrinsicoid deflection in lead  $V_6$  is normal.

*Case Summary*

Coronary arteriosclerosis. Probable subendocardial infarct. No cardiac enlargement. No congestive heart failure. Blood pressure 140/85. Radiologically, the heart is not enlarged.



One month later

A

*Explanation*

The inverted T waves in leads  $V_2$  through  $V_5$  are of the ischemic type as the ST segments are coved and the QT intervals are prolonged. The QT intervals measure 0.44 second (upper limit of normal 0.38 second - see appendix). A small anterior infarct is suspected because of the Q waves and small R waves in lead  $V_2$  which initiate the writing of the QRS complexes. Usually lead  $V_2$  is characterized by rS complexes. In this case qRS complexes are present. This is abnormal. A tracing taken two months previously showed rS complexes in lead  $V_2$  with T waves which were almost isoelectric.

*Case Summary*

Acute myocardial infarct twelve days ago. Clinically the infarct was small as the white count stayed below 12,000, the sedimentation rate only reached 26 mm/hour and the blood pressure fell only slightly.



I A

II A

*Explanation*

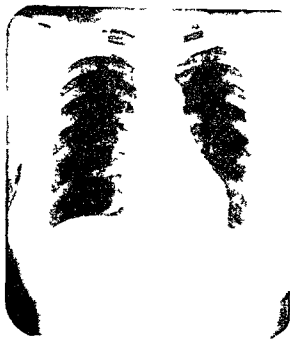
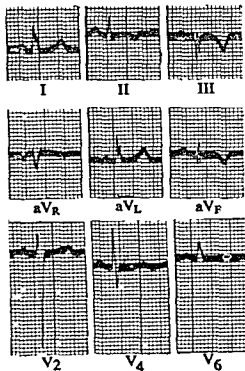
The tallest QRS complexes of the three standard limb and unipolar limb leads are 5.0 mm (lead I). The complexes are measured from the top of the base line to the top of the positive waves and from the bottom of the base line to the bottom of the negative waves and the sum of these values taken. Typically low voltage is present when the values obtained are less than 5.0 mm. Low voltage in the limb leads occurs in some normal individuals when the electric forces are moving perpendicular to the frontal plane of the body. It occurs also with edema, myocardial disease, pericardial effusion, etc.

The QRS intervals are not abnormal. The intervals may vary from 0.06 through 0.10 second in adults (see appendix).

Notching of the T waves is present in leads  $V_3$  and  $V_4$ . This occurs commonly with coronary artery disease and is present in a small percentage of normal subjects.

*Case Summary*

Coronary arteriosclerosis and angina pectoris at the time of the first tracing. Heart upper limit of normal in size. There is rounding at the apex. The cardiothoracic ratio is 0.44. Three months after this tracing the patient had severe precordial pain which lasted six hours. The tracing taken 14 days later is shown below. The inverted T waves in leads II, III, and  $aV_F$  indicate posterior myocardial ischemia while the development of Q waves in lead III and lowering of initial r waves in lead  $aV_F$  indicate a small posterior myocardial infarct. No digitalis or quinidine at the time of either tracing.



Fourteen days after prolonged severe chest pain

## Answers

I A

II A

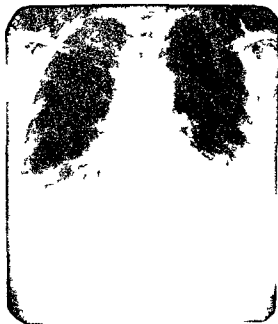
### Explanation

The R waves in leads I  $aV_L$  and  $V_6$  are tall measuring 18.0 mm, 17.0 mm, and 24.0 mm respectively (upper limits of normal 11.0 mm, 10.0 mm, and 22.0 mm respectively see appendix). The S waves are unusually deep in lead  $V_2$  measuring 34.0 mm (upper limit of normal 29.0 mm see appendix). The R/T ratios in leads I  $aV_L$  and  $V_6$  are abnormal indicating relative lowering of the T waves with respect to the amplitudes of the R waves.

A ventricular ectopic beat is present in lead  $V_1$ .

### Case Summary

Essential hypertension, ten years. Average blood pressure 220/120. Highest pressure 265/135, lowest pressure 180/110. Heart enlarged to the left. The cardiothoracic ratio is 0.59. The increased densities at both lung bases are due to pulmonary congestion and large breast shadows.



*Answer*

B

*Explanation*

Subepicardial ischemia of the anterolateral aspect of the left ventricle produces inverted T waves in the precordial leads. The electrocardiographic position of the heart is semivertical. Sinus rhythm is present with a rate of 70 beats per minute.

*Case Summary*

Coronary insufficiency with myocardial ischemia. A large hiatus hernia is present also. The negative S-T segment shifts in lead II appeared during an attack of chest pain which improved with nitroglycerin. Teleoroentgenogram shows that the stomach is posterior to the heart on the left. The cardiac size is obscured. The densities at the right lung base suggest adhesions.



Postero-anterior  
Position



Left Lateral  
Position

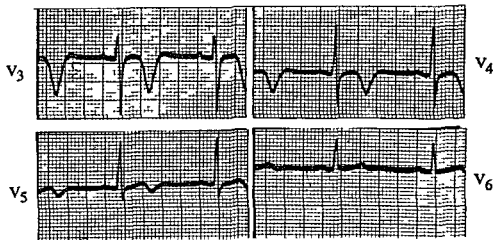
B

*Explanation*

The tracing is within normal limits however a previous electrocardiogram taken one year ago showed deeply inverted T waves in the precordial leads (see below) It is important to realize that a normal electrocardiogram does not rule out the possibility of heart disease but must be considered in the light of the history and other findings Sinus rhythm rate 74 beats per minute

*Case Summary*

Coronary arteriosclerosis coronary insufficiency and angina pectoris are the most likely diagnoses The patient has attacks of substernal burning pain on effort which radiates into the throat The average blood pressure is 150/103 Pericarditis could produce a similar electrocardiographic picture with changes in S T segments and T waves but without changes in the QRS complexes Heart upper limit of normal in size The lung fields are clear



Tracing Taken One Year Ago



*Answer*

**B**

*Explanation*

The apparent widening of the P waves in the precordial leads is due to merging of U waves and P waves. Left ventricular hypertrophy is suspected because of the R waves in leads  $V_4$ ,  $V_5$  and  $V_6$  which are at the upper limit of normal and which are associated with negative J shifts and flat S T segments.

*Case Summary*

Hypertensive heart disease, blood pressure about 190/118 for twenty years. Heart appears enlarged to the left. There is a fleck of calcium in the aortic arch. Increased density at the right lung base, probably due to pulmonary fibrosis.



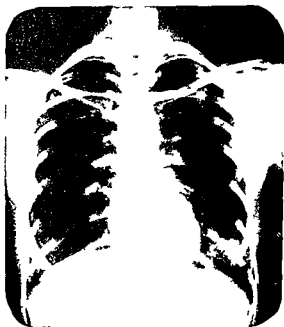
D

*Explanation*

The J points are elevated 2.0 mm in lead  $V_2$  (upper limit for normal for this lead is 1.5 mm see appendix). It is to be noted that the upper limit of normal for J is 2.0 in lead  $V_3$ . Thus a slight shift in electrode placement could alter the J point in leads  $V_2$  and  $V_3$ . Sinus rhythm rate 62 beats per minute. The notching on the upstroke of the R waves in lead  $V_2$  is unusual and represents a point in the transition from the R waves in lead  $V_1$  to the QR waves in lead  $V_3$ . This is not abnormal. There is no definite electrocardiographic evidence of cardiac disease.

*Case Summary*

No heart disease. Chronic bronchial asthma. Heart normal size, shape and position. The cardiothoracic ratio is 0.40. Lungs clear.



## Answers

I C

II B

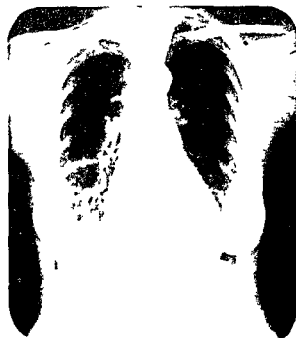
## Explanation

The R waves in lead  $V_4$  are high as they exceed the upper limit of normal which is 27.0 mm (see appendix)

Left ventricular hypertrophy is suspected because of the high R waves and flattening of the ST segments in leads  $V_4$  through  $V_6$ . Sinus rhythm rate 62 beats per minute

## Case Summary

Hypertensive heart disease Old healed pulmonary tuberculosis The heart is enlarged greatly downward and to the left Cardiothoracic ratio is 0.67 There are old scars at the right apex and base



## Answers

I C

II C

## Explanation

Rapid jumps in the base line are present in various leads. The electrode which was attached to the left foot was loose. Movement of the electrode on the skin usually produces a wandering base line rather than sharp jumps in the base line as shown in this tracing.

Left ventricular hypertrophy is present because of the high R waves, negative S-T segments and inverted T waves in leads I, V<sub>5</sub> and V<sub>6</sub>. The relative shortening of the Q-T intervals suggest a digitalis effect.

## Case Summary

The patient has had essential hypertension for many years. Highest blood pressure 250/150. Lowest 220/115. The heart is enlarged as shown by the rounding of the apex. The cardiothoracic ratio is 0.48.



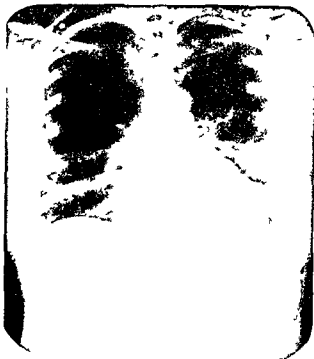
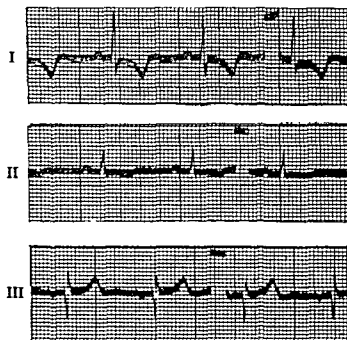


B

Left ventricular hypertrophy is suggested by the relatively deep S waves in lead  $V_3$  and J points S T segments and T waves which are discordant with the major deflections of the QRS complexes in lead  $V_6$ . Myocardial injury and ischemia can produce coved inverted T waves of this type. In this case the patient had a myocardial infarct clinically eight months ago at which time negative S T segment shifts in leads I and II and positive shifts in lead III became apparent. Seven months ago coved inverted S T segments and T waves were seen in lead I and positive T waves were present in lead III (see below). The tracing about which the questions were asked shows less negative T waves and J shifts in lead I. All of the variations occurred without significant changes in the QRS complexes. It is probable that the present tracing represents myocardial injury and ischemia as well as left ventricular hypertrophy.

### Case Summary

Hypertensive arteriosclerotic heart disease probable old myocardial infarct angina pectoris. Average blood pressure 190/105. The heart is enlarged to the left. The cardiothoracic ratio is 0.56. Calcification of the aorta is present.



Seven months ago

A

*Explanation*

Lead III has been mounted in the lead I position and lead I in the lead III position. An abnormality in mounting is suspected because the electric axis of the QRS complexes as estimated from the unipolar limb leads is 10 degrees but estimated from the standard leads is 90 degrees. Einthoven's law (the sum of the amplitudes of the QRS complexes of leads I and III equals lead II) holds true in the presence of this type of mismounting error. Thus the application of this law is not helpful in revealing this type of mounting error. The artifact is not due to switching of the right arm and left arm lead wires because such an artifact can be corrected by turning lead I upside down and interchanging leads II and III. When this is done the electric axis of the QRS complexes is not consistent with that derived from the unipolar limb leads. Lead I is not mounted upside down as turning lead I over would not correct the electric axis and because P waves precede and T waves follow the QRS complexes.

*Case Summary*

No heart disease. The heart is not definitely enlarged. The cardiothoracic ratio is 0.47. After correcting the artifact the electrocardiogram is entirely within normal limits.



## Answers

I B

II B

## Explanation

The first portion of the QRS complexes in lead  $V_6$  is nearly isoelectric. This is seen clearly when leads  $V_2$  and  $V_6$  are recorded simultaneously with a paper speed of 75 mm per second. The transitional zone falls at lead  $V_2$ . Normally it falls at  $V_2$  or  $V_3$  and rarely at lead  $V_4$ . Sinus rhythm rate 61 beats per minute. Normal electrocardiogram.

## Case Summary

Normal male. Heart normal size, shape and position.



## Answers

I C

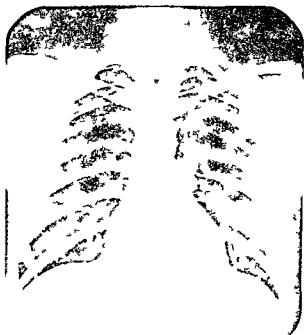
II B

## Explanation

The electric axis is 55 degrees and is derived as follows. Take the algebraic sum of the amplitudes of the positive and negative waves of the QRS complexes in lead I (this gives 8 mm). Perform the same calculation on lead III (this gives 6 mm). The electric axis is derived by using these figures in the appropriate table (see appendix). Electrocardiograms are usually abnormal if the axis deviation is more negative than minus 30 degrees or more positive than plus 102 degrees (see appendix).

## Case Summary

Normal tracing. Vasovagal attack. Heart normal size, shape and position. The cardiothoracic ratio is 0.43. Calcified lymph nodes are present in the left hilum.



## Answer

F

## Explanation.

Sinus bradycardia is present as the cardiac rate is less than 60 beats per minute and the pace maker is in the sinus node. Reserpine can cause a slowing of the heart rate such as this. The T waves tend to be low in lead II however they are not outside of normal limits.

## Case Summary

Moderate obesity. No heart disease. The heart is normal size, shape and position. The cardiothoracic ratio is 0.48.



## *Answers*

I C

II E

## *Explanation*

Notching of the T waves in the presence of long Q T intervals with U waves superimposed on the downstroke of the T waves suggest quinidine administration. The fine tremor in the limb leads is due to muscle potentials. The variable intensity of the vertical time lines suggests a loose connection in the lamp circuit. An atrial or nodal premature contraction is present in lead V<sub>6</sub>.

The electrocardiographic position is semivertical because the ventricular complexes of lead aV<sub>F</sub> resemble those of leads V<sub>5</sub> and V<sub>6</sub> and the ventricular complexes of lead aV<sub>L</sub> are small.

## *Case Summary*

Paroxysmal atrial fibrillation etiology unknown. The patient is taking quinidine sulfate 0.8 gm (12 gr) daily.

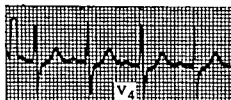
B

*Explanation*

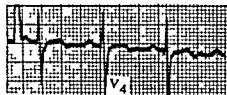
Wide QRS complexes of ventricular origin which are not preceded by P waves occur periodically. The QRS complexes occur early in time and are therefore premature contractions. Sinus rhythm rate 76 beats per minute. The slight negative ST segment shifts in leads  $V_4$  and  $V_5$  are suggestive but not diagnostic of cardiac disease. Later tracings show negative ST segments in lead  $V_4$  on 12/27/55 and inverted T waves on 12/28/55 (see below). The P waves following QRS complexes in leads III and  $aV_L$  are due to retrograde conduction (from ventricles to atria).

*Case Summary*

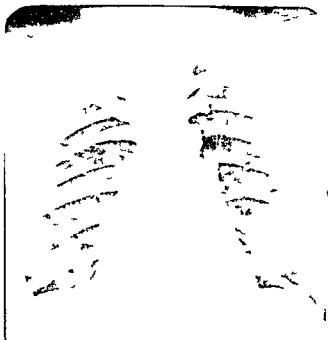
Coronary arteriosclerosis. A subendocardial infarct was diagnosed because of the severe chest pain of long duration, rapid sedimentation rate, elevated serum transaminase and serial changes in the electrocardiogram. The heart is not enlarged. The cardiothoracic ratio is 0.39. The great vessel shadow is enlarged slightly. There are densities in the lower lung fields due to lipomas in the subcutaneous tissue.



12/27/55



12/28/55



### Answer

B

### Explanation

An anterior infarct is present because of the large Q waves in leads  $V_3$ ,  $V_4$  and  $V_5$ . The intrinsicoid deflection is delayed in lead  $V_6$ . The infarct is subacute as there are positive ST segment shifts in leads  $V_3$ ,  $V_4$  and  $V_5$  which are associated with deeply inverted T waves. The ST segments are coved. In addition the Q waves in leads II, III and  $aVF$  are suggestive of posterior wall infarction. Sinus tachycardia rate 107 beats per minute.

### Case Summary

Myocardial infarct five weeks previously. There was no history to suggest two separate myocardial infarcts. Thus it is possible that the anterior infarct reduced the circulation to the posterior wall through vasoconstriction which resulted in infarction of the posterior wall of the left ventricle. Radiographically the heart was not enlarged.



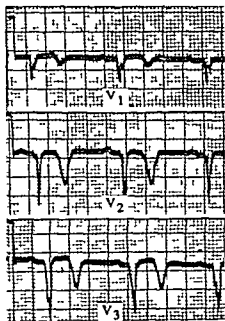
A

*Explanation*

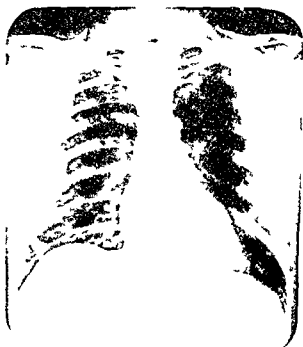
Q waves are present in leads  $V_1$  and  $V_2$  which suggest the presence of an anterior myocardial infarct. Usually R waves initiate the writing of the QRS complexes in these leads. The delayed intrinsicoid deflections in lead  $V_1$  which measures 0.04 second (upper limit of normal 0.03 second see appendix) may be due to the infarct also. Myocardial ischemia adjacent to the infarct is suggested by the inverted T waves in leads  $V_2$  through  $V_4$ . It cannot be said that an infarct is certain from the tracing as myocardial death due to tumor, trauma, gumma, Ashoff bodies, etc. would produce similar findings. Sinus rhythm, rate 75 beats per minute.

*Case Summary*

Myocardial infarct one week ago. Deep QS waves in leads  $V_1$ ,  $V_2$  and  $V_3$  developed nine days later which suggested the presence of a larger area of myocardial necrosis (see below). Heart upper limit of normal in size. The cardiothoracic ratio is 0.44. The lung fields are clear.



Nine days later



C

*Explanation*

Large Q waves are present in leads II, III and  $aV_F$ . In lead  $aV_F$  the Q/R ratios are 1.2 (they should not exceed 0.28 see appendix). The Q/R ratios are still abnormal after inspiration, the ratios being 0.50. The Q waves in lead  $aV_F$  are greater than 0.04 second in duration. Q waves which are 0.04 second or greater usually indicate posterior myocardial infarction. It has been stated that a posterior infarct is likely when in lead  $aV_F$  the Q waves measure 0.03 second or more from the onset of the QRS complexes to the nadir of the waves with Q/R ratios which are greater than 0.25 when the amplitudes of the QRS complexes are greater than 5.0 mm. Sinus tachycardia rate 101 beats per minute.

*Case Summary*

Coronary arteriosclerosis. Old myocardial infarct. Heart normal size, shape and position. The cardiothoracic ratio is 0.43.



## Answers

I B

II B

III B

## Explanation

Sinus arrhythmia is present because there is phasic variation of the cardiac rate with respiration. This is shown clearly in leads  $V_2$ ,  $V_3$  and  $V_6$ . In lead  $V_6$  the height of the waves also changes with respiration.

The electrocardiogram is within normal limits. The R waves in lead  $V_3R$  and  $V_1$  are consistent with the right ventricular predominance which is normal at an early age. Also the inverted T waves in leads  $V_4R$ ,  $V_3R$  and  $V_1$  are normal for this age. It is not unusual for T waves to be inverted in leads  $V_2$ ,  $V_3$  and  $V_4$  in children (see appendix).

The wandering base line is due probably to movement of the electrode on the skin. A loose electric connection causes abrupt shifts in the base line.

## Case Summary

Normal child. Radiologically the heart is normal size, shape and position.



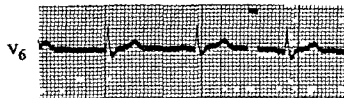
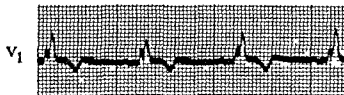
## A

## Explanation

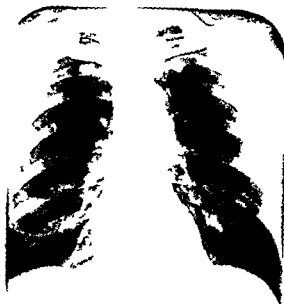
Sinus bradycardia is present because the rate is below 60 beats per minute being 55 beats per minute. A complete bundle branch block is present because the duration of the QRS complexes is 0.12 second or greater (in lead I it measures 0.13 second). The block involves the right bundle of His because the terminal waves of the QRS complexes are prolonged and are predominantly positive in the tracings recorded from regions adjacent to the right ventricle and negative from regions adjacent to the left ventricle. The intrinsicoid deflections in lead  $V_1$  are delayed measuring 0.09 second (upper limit of normal is 0.03 second see appendix). The electrocardiographic position is semivertical because the complexes of lead  $aV_L$  are small and those of lead  $aV_F$  resemble those of leads  $V_5$  and  $V_6$ . The tracing has not changed significantly over a two year period (see below). Thus the tracing represents probably a block in the right bundle of His without significant disease elsewhere in the myocardium.

## Case Summary

Essential benign hypertension highest blood pressure 204/130 lowest blood pressure 168/88. Radiologically the descending aorta is curved and the heart is not enlarged. The cardiothoracic ratio is 0.31. Coronary arteriosclerosis may be the etiology of the right bundle branch block however a right bundle branch block may occur with only minor changes in the myocardium for example with slight increases in pressure in the right ventricle.



Tracing taken 2 years ago



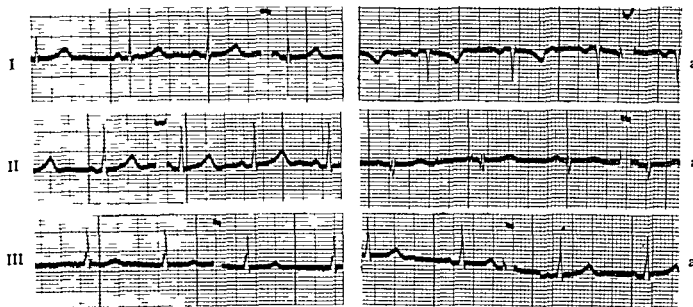
C

*Explanation*

The lead marked  $aV_R$  appears to be recorded correctly as it is consistent with the precordial leads and in general resembles lead  $V_1$  and is the mirror image of lead  $V_6$ . This suggests that the lead wire of  $aV_R$  was placed properly on the right arm and therefore the error must have been made when applying the lead III electrodes to the left arm and leg. The standard leads may be corrected for this type of error by interchanging leads I and II and viewing lead III as the mirror image. The unipolar limb leads may be corrected by interchanging leads  $aV_L$  and  $aV_F$ . The tracing recorded properly is shown below. Semivertical electrocardiographic position.

*Case Summary*

Normal female. No evidence of cardiac disease. Radiologically the heart is normal size, shape and position.



Limb Leads Recorded Correctly

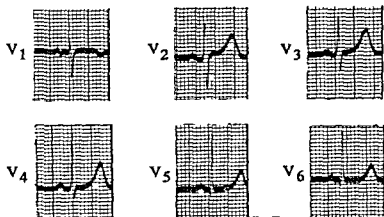
C

*Explanation*

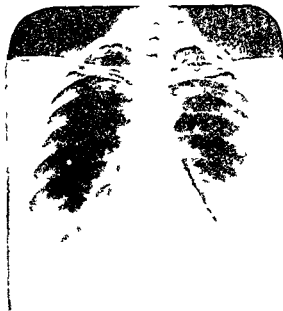
Leads  $aV_F$  and  $V_1$  through  $V_6$  are all similar. Thus the precordial leads were recorded with the lead selector switch in the  $aV_F$  position. The tracing is normal otherwise. The electrocardiographic position is semihorizontal.

*Case Summary*

Normal female. Teleoroentgenogram shows the heart to be normal size, shape and position. The aortic knob is prominent. The cardiothoracic ratio is 0.42.



Precordial leads recorded correctly

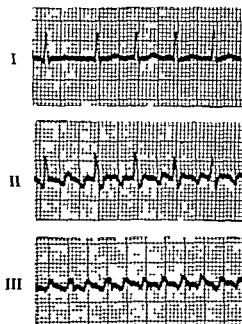


*Explanation*

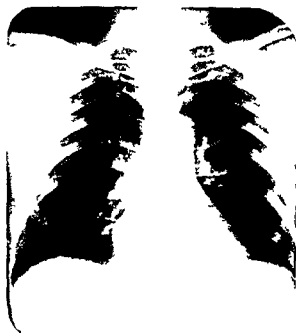
Atrial premature contractions with aberration of the QRS complexes are present as certain P waves occur early in time and are followed by wide QRS complexes with secondary T wave changes. A premature atrial beat shows clearly in lead V<sub>1</sub>. The QRS complexes following the premature beats resemble those seen in a right complete bundle branch block. Thus it is probable that these complexes represent a delay in the conduction in the right bundle of His. Aberration following atrial premature beats shows right delay or block in a large percentage of cases. The compensatory pause is incomplete (the sum of the time before and after the premature beat is less than that between two atrial contractions which are initiated from the SA node). Atrial premature contractions may be caused by atrial disease due to rheumatic fever, arteriosclerosis, hyperthyroidism or to anxiety, caffeine or other causes. The tracing is otherwise within normal limits.

*Case Summary*

No definite evidence of cardiac disease could be found and all of the causes listed above were searched for. A tracing taken three months later showed typical atrial flutter with a 2:1 block. The teleoroentgenogram is normal. The cardiothoracic ratio is 0.47. The blood pressure was 130/90. (See below.)



3 months later



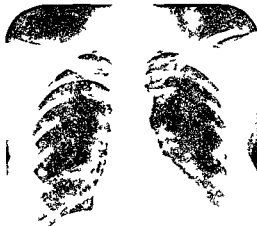
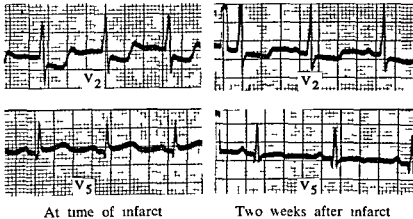
C

### Explanation

An old posterior or posterolateral myocardial infarct could be present with a tracing of this sort. With posterior infarction there is a tendency for the T waves to become lower and inverted in lead I and positive in lead III. Also a posterior infarct destroys forces on the posterior aspect of the heart which leaves an overbalance of forces on the right and gives rise to tall R waves in lead V<sub>1</sub>. In this tracing the R waves are relatively tall in lead V<sub>1</sub> with an abnormal R/S ratio. Although the tracing is consistent with a diagnosis of a posterior infarct, this diagnosis cannot be made with certainty. A tracing taken at the time of the precordial pain and two weeks later showed negative ST segment shifts in leads V<sub>2</sub> and V<sub>3</sub> which are consistent with a diagnosis of a myocardial infarct.

### Case Summary

Clinically the patient had a typical myocardial infarct with shock. The heart is enlarged to the left. The cardiothoracic ratio is 0.59.



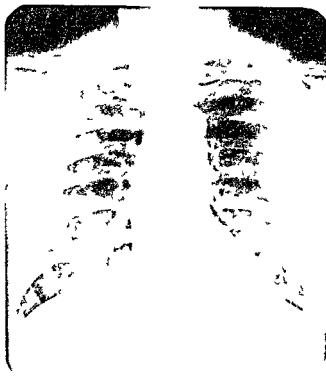
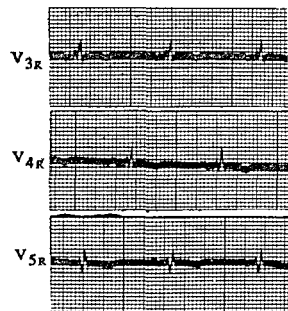


### Explanation

An incomplete right bundle branch block is suspected because of the wide R waves in lead  $aV_R$  high notched QRS complexes in lead  $V_1$  delayed intrinsicoid deflection in lead  $V_1$  and relatively wide S waves in lead  $V_6$  and other leads. Additional leads taken to the right support this diagnosis because of the presence of R R prime waves. Usually with an incomplete right bundle branch block the intrinsicoid deflection in lead  $V_1$  is from 0.05 to 0.075 second. The coving of the S T segments in lead  $V_1$  represents the transition between the positive T waves in lead  $V_2$  and the negative T waves in leads  $V_3R$ ,  $V_4R$  and  $V_5R$ . It is important to point out that often right ventricular hypertrophy, incomplete and complete right bundle branch block are difficult to differentiate. The time of onset of the intrinsicoid deflection is helpful but is somewhat arbitrary and based on relatively few cases and thus should not be relied upon as the sole finding when making this differentiation.

### Case Summary

The patient has essential hypertension, highest blood pressure 220/120, lowest pressure 180/108. There is no definite history of angina pectoris. Teleoroentgenogram of the chest shows that the heart is not enlarged in its transverse diameter. The great vessel shadow is widened slightly. It is probable but not certain that the incomplete right bundle branch block is the result of coronary artery disease.



**B**

*Explanation*

A right complete bundle branch block is present as the QRS complexes in lead III measure 0.13 second (upper limit of normal 0.10 second see appendix) and there are tall late R waves in lead  $V_1$  which measure 10 mm (upper limit of normal is 7 mm). Wide S waves are present in lead  $V_6$ . The intrinsicoid deflections in lead  $V_1$  exceed the upper limit of normal of 0.03 second measuring 0.09 second. A right complete bundle branch block usually is characterized by an intrinsicoid deflection which is greater than 0.08 second. An anteroseptal infarct is present because of the Q waves in lead  $V_3$  with elevated ST segments and inverted T waves in leads  $V_1$  through  $V_4$ . There is a small wide q wave in lead  $aV_F$ . Sinus tachycardia is present with a rate of 101 beats per minute.

*Case Summary*

Acute myocardial infarction and right complete bundle branch block. It is possible that the infarct involves the septum and the right bundle of His as well.

## Answers

I B

II B

### Explanation

The P pulmonale pattern is present that is the P waves in lead I are small and are tall and peaked in leads II, III and aV<sub>F</sub>. This suggests right atrial enlargement but may be simulated by cardiac rotation in subjects with a low diaphragm. Small r and deep S waves occur in most precordial leads. These suggest that the precordial electrodes are facing the right ventricle probably because of strong clockwise rotation of the heart about the long axis as viewed from the apex.

### Case Summary

Pulmonary emphysema typical. Sinus tachycardia rate 102 beats per minute. The heart is small and in the midline. The cardiothoracic ratio is 0.34. The diaphragms are flat and low. The ribs are horizontal. The decreased pulmonary markings especially at the right lung base with increased densities at the upper lung fields indicate emphysema and pulmonary fibrosis.



### *Answer*

**B**

### *Explanation*

The sagging of the S T segments in leads V<sub>4</sub> V<sub>5</sub> and V<sub>6</sub> and relatively short Q T intervals suggest digitalis administration. The tracing is in keeping with but is not diagnostic of mitral stenosis. The width of the P waves is 0.12 second which is prolonged slightly and the P waves are flat topped in leads I V<sub>5</sub> and V<sub>6</sub>. The P R intervals are at the upper limit of normal being 0.20 second in lead II. Digitalis, rheumatic heart disease, myocardial ischemia and other states may prolong the P R interval.

### *Case Summary*

Rheumatic heart disease with mitral stenosis and insufficiency. The mitral orifice was 1.0 cm before fracture of the valve. Phonocardiogram from the mitral area one year after surgery showed an early diastolic decrescendo and late crescendo murmur.

B

*Explanation*

An anteroapical infarct is present because of the QS waves in lead  $V_3$ . The infarct is subacute as the ST segments are isoelectric and are associated with deeply inverted and coved T waves in leads  $V_3$  and  $V_4$ . It is significant that r waves are present in leads  $V_1$  and  $V_2$  but are absent over the infarct in lead  $V_3$ . It should be pointed out that although tracings of this sort are called subacute one cannot estimate the duration of the infarct consistently from a single tracing. Sinus rhythm rate 78 beats per minute. There is a tendency toward low voltage of the QRS complexes in the limb leads.

*Case Summary*

Myocardial infarct seven weeks prior to this tracing. Heart normal size, shape and position. The cardiothoracic ratio is 0.44. The aortic knob is somewhat prominent.



*Answer*

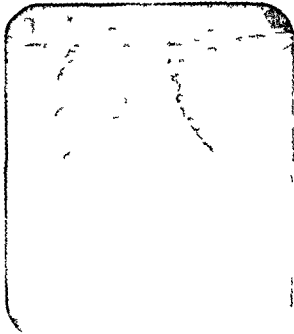
A

*Explanation*

Atrial fibrillation and a fine muscle tremor are present. Hyperthyroidism is a frequent cause of these abnormalities when they exist together. The sagging of the S-T segments and short Q-T intervals suggest digitalis administration.

*Case Summary*

Hyperthyroidism. BMR plus 45 and blood iodine 12 mcgm per cent. The heart is enlarged slightly to the left. The cardiothoracic ratio is 0.49.



## *Answers*

I C

II D

### *Explanation*

Ventricular tachycardia is present as shown by the wide QRS complexes with secondary type T waves. The R-R intervals are somewhat irregular which is characteristic of this condition. The diagnosis of ventricular tachycardia can generally be made with certainty when independent behavior of the atria and ventricles can be demonstrated by detecting the presence of occasional P waves superimposed on the ventricular complexes. P waves superimposed on QRS complexes can be detected in tracing by using calipers.

A quinidine effect is suggested from tracing F because of the long Q-T intervals. Digitalis and hypercalcemia characteristically shorten Q-T intervals. The effect of hyperkalemia on the Q-T intervals usually is not great.

### *Case Summary*

Hypertensive arteriosclerotic heart disease. Highest blood pressure 185/115. Lowest pressure 160/98. No cardiac enlargement or congestive heart failure. The ventricular tachycardia stopped after the intravenous administration of 0.7 gm of quinidine in two hours.

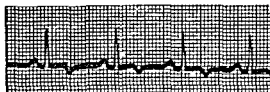
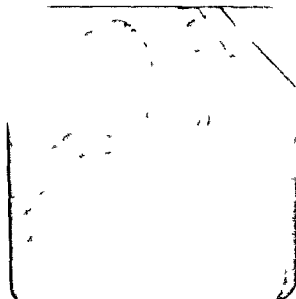
D

*Explanation*

The inverted T waves in the presence of definitely positive QRS complexes in leads aV<sub>F</sub>, V<sub>2</sub> and V<sub>6</sub> are consistent with a diagnosis of chronic pericarditis. These changes suggest involvement of the posterolateral and diaphragmatic surfaces of the left ventricle which is consistent with the locations of the areas of calcification shown in the X rays below. The T wave changes are chronic as no significant changes have occurred in the last three years (see below). The relatively tall R waves in lead V<sub>5</sub> are in keeping with a diagnosis of left ventricular hypertrophy shown in the X rays. The P waves in leads II and III are wide and notched. These findings are not uncommon in patients with constrictive pericarditis especially if the constriction occurs around the AV groove.

*Case Summary*

Chronic adhesive pericarditis of uncertain etiology. Left ventricular hypertrophy. The cardiothoracic ratio is 0.47. Calcification and adhesions to the pericardium are present. Twenty five years ago the patient had what was diagnosed as rheumatic heart disease with pericardial effusion. No cardiac murmur. No evidence of valvular disease.

Lead V<sub>6</sub> three years agoPartial left anterior  
oblique view

Left lateral view



I A

II B

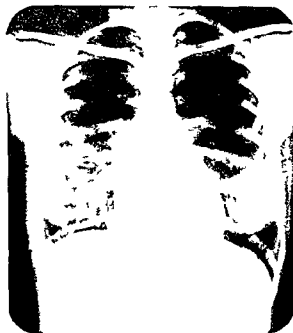
*Explanation*

Sinus arrest is shown in leads I II  $V_1$  and  $V_4$ . Sinus arrest is a temporary cessation of sinus node activity due often to increased vagal tone or to depressant drugs. The P R intervals vary in length due perhaps to varying rates of AV conduction. It is probable that the drugs were additive in their effects, all acting to depress cardiac activity. A diagnosis of incomplete sino-atrial block was not made because in this condition the periods of cardiac standstill are almost exact multiples of the normal heart cycles. This was not the case in this tracing.

A digitalis effect is present because of the saucer shaped S T segments with relatively short Q T intervals. Reserpine has no significant effect on the electrocardiogram except for the bradycardia which it may produce. Quinidine lengthens Q T intervals.

*Case Summary*

Toxic heart disease due to drugs was present. Possible rheumatic heart disease. There was an apical grade I systolic murmur but no diastolic murmur. Fluoroscopically the left atrium was enlarged slightly. The syncopal attacks disappeared when the drugs were stopped. X ray showed a cardiothoracic ratio of 0.46. Old linear scars were present at the right lung base.



## *Answer*

C

## *Explanation*

The P R intervals measure 0.40 second (upper limit of normal 0.20 second see appendix) A posterior myocardial infarct is indicated in lead aV<sub>F</sub> by the Q waves which measure 0.04 second in duration and the Q/R ratios which are 0.60 (upper limit of normal 0.28) The infarct is acute because of the elevation of the S T segments in lead aV<sub>F</sub> and abnormal segments in other leads Left ventricular hypertrophy is present because of the high voltage of the R waves in lead V<sub>5</sub> which measure 48.0 mm (upper limit of normal 26.0 mm see appendix) Digitalis effect is shown by the saucer shaped S T segments of typical configuration in leads V<sub>5</sub> and V<sub>6</sub> which are diphasic and are of the minus plus type and by the small terminal portions of the T waves

## *Case Summary*

Essential hypertension for ten years with radiographic evidence of left ventricular hypertrophy  
The acute posterior myocardial infarct occurred two days previously

## Answers

I A

II B

## Explanation

Atrial fibrillation is present because of the absolute irregularity of the R R intervals and absence of clearly distinguishable P waves preceding each QRS complex. The semivertical to vertical electrocardiographic position of the heart in the presence of atrial fibrillation is suggestive of rheumatic mitral stenosis.

A digitalis effect is present as shown by the typical saucer shaped S T segments and short Q T intervals which measure 0.32 second in lead V<sub>5</sub>. The lower limit of normal for a cycle length of 1.30 seconds is 0.38 second (see appendix). The quinidine in this case apparently did not prolong the Q T intervals significantly.

## Case Summary

Rheumatic mitral stenosis. Left atrial enlargement. Grade II apical diastolic murmur. No systolic murmur. Atrial fibrillation. The heart shows a mitral configuration with fullness in the region of the left atrial appendage. The walking point is displaced toward the apex. The right ventricular border is rounded. The cardiothoracic ratio is 0.56.



## Answers

I A

II A

## Explanation

Atrial premature contractions are present because the P waves of the ectopic beats appear prematurely and the QRS complexes are like those of the basic mechanism. The P waves of the ectopic beats differ in size and form from those of the basic mechanism and the P R intervals are shorter. The compensatory pause is incomplete.

Certain features of the tracing are suggestive of left ventricular hypertrophy. The amplitude of the R waves in lead I is at the upper limit of normal and the T waves in this lead are relatively low. There is a tendency toward left axis deviation of the QRS complexes (3 degrees) with a normal axis of the T waves (49 degrees). In lead V<sub>6</sub> there is slight sagging of the S-T segments and T waves which are low with respect to the height of the R waves. (The R/T ratios are slightly abnormal see appendix)

## Case Summary

Essential benign hypertension. highest blood pressure 250/120. lowest pressure 200/98. Exogenous obesity. height 62.5 inches. weight 185 pounds. The heart is enlarged to the left with a cardiothoracic ratio of 0.50.



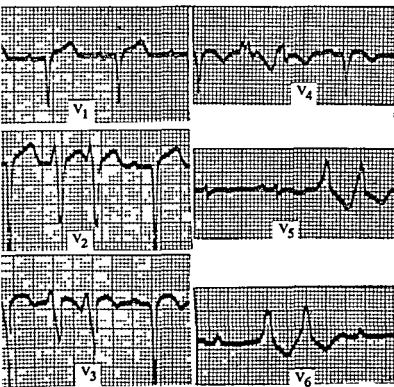
## B

*Explanation*

The tracing suggests quinidine effect but is essentially normal otherwise. Long Q T intervals are present which measure 0.44 second in lead I. According to Ashman's chart (see appendix) the upper limit of normal for Q T intervals for a rate of 71 beats per minute (cycle length 0.85 second) is 0.38 second. The Q T interval corrected for heart rate using the Bazett formula (see appendix) is 0.48. The upper limit of normal for adults is 0.44 (see appendix). The notching of the T waves in leads  $V_2$  and  $V_3$  occurs commonly with quinidine. Sinus rhythm rate 71 beats per minute.

*Case Summary*

The patient has paroxysmal ventricular tachycardia and has been taking 1.6 gm (24 gr) of quinidine daily for one year. A tuberculous kidney was removed ten years ago. Heart normal size, shape and position. The cardiothoracic ratio is 0.40. The tracing remained essentially unchanged for five years except for four attacks of typical ventricular tachycardia after which an anterior myocardial infarct occurred (see below).



Before Infarct

Two months after the  
Anterior Infarct

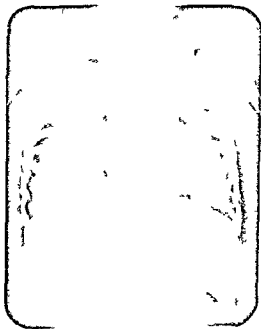
A

*Explanation*

Right ventricular hypertrophy is present. The R/S ratios are abnormal in lead  $V_1$  which indicate the presence of tall R waves with respect to the depth of the S waves. There is right axis deviation of the QRS complexes. Septal hypertrophy is suggested by the large Q waves in leads  $V_5$  and  $V_6$ . Also S waves are present in these leads which is a common finding with right ventricular hypertrophy. Deep S and Q waves in leads  $V_5$  and  $V_6$  are common with ventricular septal defects either pure or complicated by other congenital anomalies.

*Case Summary*

Tetralogy of Fallot operated on successfully. Teleoroentgenogram of the chest taken before surgery shows the apex of the heart lifted from the diaphragm indicating right ventricular hypertrophy. The pulmonary conus area is not prominent and there is a decrease in the pulmonary vascular markings suggesting decreased pulmonary blood flow.



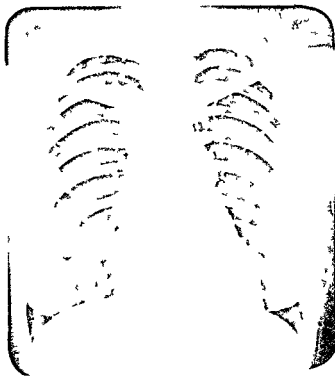
D

*Explanation*

Anomalous atrioventricular conduction is suggested by the short P R intervals and wide QRS complexes. During this time of recording of leads I, II and  $V_6$  changes in the QRS complexes and T waves occurred. The QRS complexes associated with large areas are followed by large discordant T waves which are of the secondary type. The cause of the anomalous atrioventricular conduction is not clear. Certain cases may be due to an accessory muscle bundle (Bundle of Kent) connecting atria and ventricles. Other cases may be due to an irritable focus in the AV node which produces early excitation and depolarization of the ventricles. This is followed soon after by a normal ventricular depolarization process which results in a fusion of ventricular beats.

*Case Summary*

The patient has rheumatic mitral stenosis and insufficiency with an apical grade II systolic and grade I diastolic murmur. The X ray shows fullness in the region of the left atrial appendage.



C

*Explanation*

A posterior infarct is present. The Q waves in lead aV<sub>F</sub> are suggestive of posterior infarction as the Q/R ratios remain abnormal even with deep inspiration. Sinus rhythm rate 70 beats per minute.

*Case Summary*

Posterior myocardial infarction one year ago. The heart is of normal size, shape and position. The cardiothoracic ratio is 0.34. Calcified lymph nodes are present in the left hilum.





## Answers

I B

II B

## Explanation

The electrocardiographic position is semivertical. The ventricular complexes of lead  $aV_F$  resemble those of leads  $V_5$  and  $V_6$  and the QRS complexes of lead  $aV_L$  are small.

The inverted T waves in lead  $aV_F$  in the presence of clearly positive QRS complexes are strongly suggestive of ischemia involving the posterior wall of the left ventricle. A tracing taken three weeks later showed flat S T segments and T waves in lead  $aV_F$  (see below).

## Case Summary

Essential hypertension, coronary arteriosclerosis with coronary insufficiency and angina pectoris. Heart normal size, shape and position. The cardiothoracic ratio is 0.37.



$aV_F$

Three weeks after previous tracing

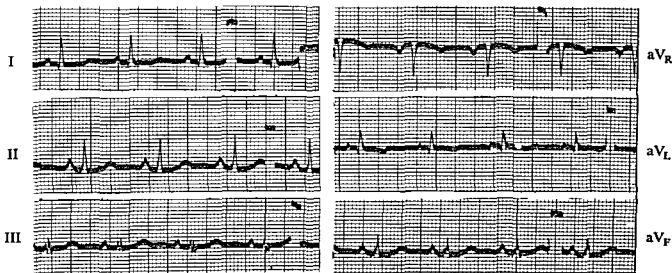


*Explanation*

A technical abnormality is present because the standard and unipolar limb leads are not consistent with the unipolar chest leads. The tracing recorded from the right arm (lead  $aV_R$ ) should if recorded properly resemble approximately lead  $V_1$  because both of these leads are facing the right side of the heart. Quite often the mirror image of lead  $aV_R$  resembles lead  $V_6$  however in this tracing the lead in the  $aV_F$  position is the mirror image of lead  $V_6$ . This suggests that the lead wire labeled left leg was placed on the right arm. From this it would appear that a switching of the lead II electrodes might have occurred (the lead wire labeled left leg was placed on the patient's left arm and the wire labeled right arm was placed on the patient's left leg). The errors produced by this abnormality can be corrected in the standard leads by forming the mirror image of each of the standard leads and interchanging leads I and III. After these changes have been made the electric axis derived from the standard leads are consistent with the unipolar limb leads and the complexes of the limb leads are consistent with those of the precordial leads. The tracing recorded correctly is shown below. The corrected electrocardiogram shows certain abnormalities. The T waves in leads  $V_2$ ,  $V_3$  and  $V_4$  are inverted or notched and indicate subepicardial ischemia of the anterior portion of the left ventricle.

*Case Summary*

Coronary arteriosclerosis Subendocardial infarct two months ago



Tracing Recorded Correctly

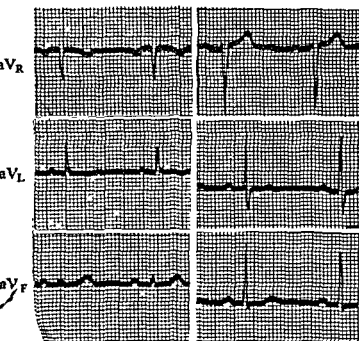
A

*Explanation*

Inverted T waves in leads  $V_3$  and  $V_4$  are produced often by myocardial ischemia. Other states such as drugs, ventricular hypertrophy, pericarditis, and electrolyte disturbances may produce abnormal T waves also. The slight sagging of the ST segments in leads I,  $V_5$ , and  $V_6$  also are abnormal and are consistent with myocardial injury or ischemia. The inverted T waves in lead  $aV_L$  also are abnormal. Often it is difficult to differentiate left ventricular hypertrophy from myocardial ischemia. The T wave changes and ST segment shifts were of sudden onset which is consistent with myocardial ischemia. The tracing taken one month ago shown below is more normal.

*Case Summary*

Arteriosclerotic coronary artery disease. Coronary insufficiency with myocardial ischemia and angina pectoris. Blood pressure has never been over 130/80 and averages 120/75. The heart is normal size, shape, and position. The great vessel shadow is widened at the base of the heart. The descending aorta is prominent.



Tracing taken 1 month previously



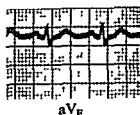
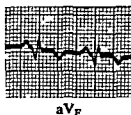
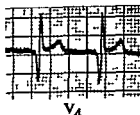
B

*Explanation*

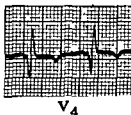
The large Q waves in leads  $V_3$  through  $V_6$  indicate the presence of an infarct involving anterior, lateral and posterolateral aspects of the left ventricle. The wide Q waves (0.04 second) in lead  $aV_F$  with Q/R ratios of 0.40 (upper limit of normal 0.28 see appendix) indicate the presence of posterior myocardial infarct. Both infarcts appear old. Large Q waves in lead  $V_4$  gave evidence of the anterior infarct four years ago and the Q waves in lead  $aV_F$  gave evidence of the posterior infarct two years ago (see below). The intrinsoid deflections in lead  $V_6$  are delayed to 0.06 second (upper limit of normal is 0.05 second see appendix). Sinus rhythm rate 99 beats per minute.

*Case Summary*

Anterior myocardial infarct four years ago and a posterior myocardial infarct two years ago. Both infarcts were distinct both clinically and electrocardiographically. The heart is essentially normal size, shape and position. The lungs are clear. The cardiothoracic ratio is 0.45.

 $aV_F$  $aV_F$  $V_4$ 

4 years ago

 $V_4$ 

2 years ago

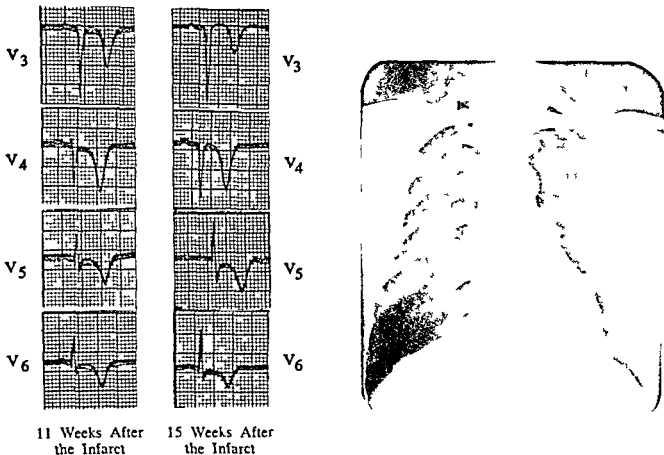


### Explanation

The QS waves in leads  $V_3$  and  $V_4$  indicate necrosis of the anterior aspect of the left ventricle. Atrial premature contractions are shown in lead  $V_3$  and other leads. The P waves of the premature beats differ in configuration from those of the basic mechanism. The QRS complexes and T waves of the premature beats are similar to those of the basic mechanism. The infarct is subacute because of the nearly isoelectric ST segments and deeply inverted T waves in the precordial leads with the abnormal Q waves. The return of the negative T waves toward normal was unusually slow in this case (see below). Thus the age of the infarct could not be determined accurately from a single tracing. Sinus bradycardia is the basic mechanism with a ventricular rate of 54 beats per minute.

### Case Summary

Arteriosclerotic coronary artery disease. Myocardial infarction. The size of the heart is within normal limits. There is a fleck of calcium in the aorta. The cardiothoracic ratio is 0.46.



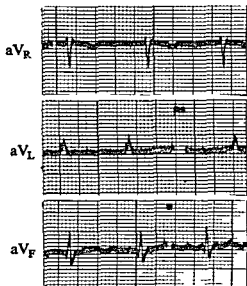
B

*Explanation*

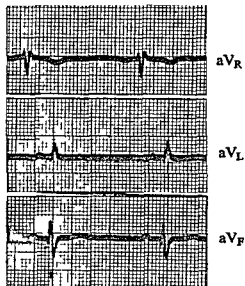
Typical flutter waves are seen in leads II III and aV<sub>F</sub>. The atrial rate is 280 beats per minute and the ventricular rates varies from 60 to 100 beats per minute. A variable AV block is present. A posterior infarct is possible because of the Q waves in leads II III and aV<sub>F</sub> but cannot be diagnosed with certainty as these waves are small.

*Case Summary*

The diagnosis is hypertensive arteriosclerotic cardiovascular disease. Large doses of digitalis were given which converted the flutter to atrial fibrillation (see below). Two days after the occurrence of atrial fibrillation quinidine was given which converted atrial fibrillation to sinus bradycardia (see below). These are typical reactions to these drugs.



After Digitalis



After Quinidine



I B

II B

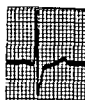
*Explanation*

The electric axis is minus 35 degrees (limit of normal minus 30 degrees, see appendix) This was obtained as follows The difference in amplitude between the Q and R waves in lead I is plus 10 mm and between the R and S waves in lead III is minus 11 mm These figures are used for determining the axis from the tables in the appendix

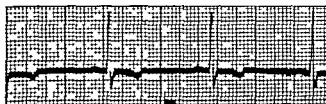
The tracing is abnormal because of the low notched T waves in lead  $V_5$  which suggest myocardial ischemia however left ventricular hypertrophy can produce changes of this sort A tracing taken seven days later showed more normal T waves which is evidence that the R wave changes were on an ischemic basis (see lead  $V_3$   $V_4$  and  $V_5$  below) Twelve days later the T waves were again inverted (see lead  $V_5$  below)

*Case Summary*

Hypertensive heart disease Highest blood pressure 228/136 lowest pressure 149/105 Coronary arteriosclerosis and insufficiency myocardial ischemia and angina pectoris Heart normal size shape and position The cardiothoracic ratio is 0.44 There is slight widening of the great vessel shadow

 $V_3$  $V_4$  $V_5$ 

Tracing 7 days later

 $V_5$ 

Tracing 12 days later



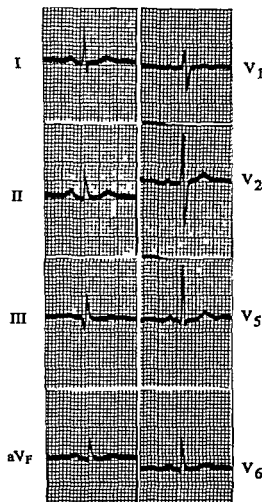
A

*Explanation*

An old posterior and posterolateral myocardial infarct is present. Large Q waves (0.04 second) are present in lead  $aV_F$ . Lead  $V_2$  recorded at 75 mm per second simultaneously with lead  $aV_F$  is of assistance in measuring accurately the width of these waves. The Q/R ratios in this lead are 2.0 and exceed the upper limit of normal which is 0.28 (see appendix). The Q waves remained unchanged in depth after inspiration which suggests a diagnosis of a posterior infarct. The inverted T waves in lead II, III,  $V_5$  and  $V_6$  suggest ischemia of the posterolateral wall of the left ventricle. The R waves in lead  $V_6$  are lower than normal and measure 1.0 mm (lower limit of normal is 4.0 mm see appendix). The tracing taken one year before the infarct shows lower R waves in lead  $V_1$  and taller R waves in leads II, III,  $aV_F$  and  $V_6$ . Sinus rhythm, rate 62 beats per minute.

*Case Summary*

Hypertensive cardiovascular disease. Myocardial infarction four months ago. Average blood pressure before the infarct 148/100. Heart enlarged to the left. The lung fields are clear.



One year  
before the infarct



I C

II A

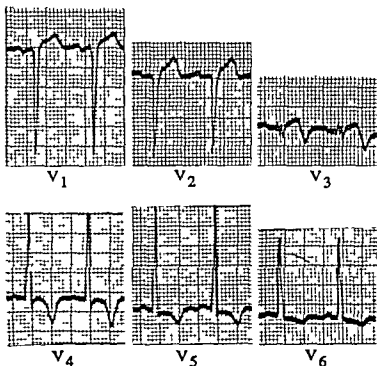
*Explanation*

A 2:1 AV block is present. There are two P waves for each QRS complex. The atrial rate is 90 and the ventricular rate is 45 beats per minute. The P waves occur regularly and are not premature in time.

The electrocardiographic position of the heart is semivertical as the ventricular complexes of lead aVL are small and the ventricular complexes of lead aVF resemble those of lead V6. Left ventricular hypertrophy is suggested by the deep S waves in lead V1 and V2 and tall R waves in leads V5 and V6 along with typical changes in the ST segments and T waves. QS waves are present in leads V1 and V2 and low R waves are present in lead V3. Left ventricular hypertrophy may account for these findings; however, an old anterior infarct could produce such changes. One year previously this patient had an attack of precordial pain of long duration associated with a rapid sedimentation rate. The tracing shown below suggests an anterior infarct. Thus the low R waves in lead V3 probably are the result of infarction. A digitalis effect is present as shown by the short QT intervals which measure 0.32 second (lower limit of normal for a cycle length of 1.32 second is 0.38 second, see appendix).

*Case Summary*

Hypertensive heart disease for fifteen years. Old myocardial infarct. Average blood pressure 185/118. The heart is not enlarged in its transverse diameter. The cardiothoracic ratio is 0.39.



Tracing taken six months ago

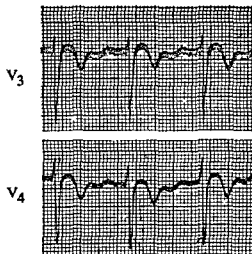
C

*Explanation*

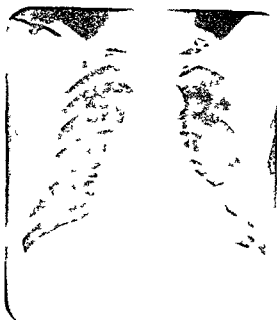
Persistent elevation of the S T segments in the precordial leads suggests a ventricular aneurysm. A tracing taken four months later revealed S T segments which were no longer elevated (see below). An acute anteroseptal myocardial infarct is suggested by the QS waves, elevated S T segments and inversion of T waves in leads  $V_2$  and  $V_3$ . A posterior myocardial infarct is suspected because of the QS waves in leads II, III and  $aV_F$ . Left ventricular hypertrophy or a horizontal electrocardiographic position in a normal subject is associated sometimes with QRS waves in lead  $aV_F$ . A first degree AV block is present as the P R intervals are 0.24 second (upper limit of normal is 0.20 second see appendix).

*Case Summary*

Myocardial infarct two days ago. No past history of a previous infarct. Aortic regurgitation of unknown etiology. The heart is enlarged to the left. There is fullness at the base of the aorta and the aortic knob is prominent. There is calcification of the aorta. The cardiothoracic ratio is 0.50. No radiologic evidence of a ventricular aneurysm.



Tracing taken four months later



I A      II B      III A

**Explanation**

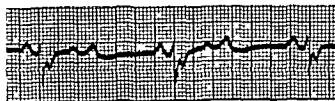
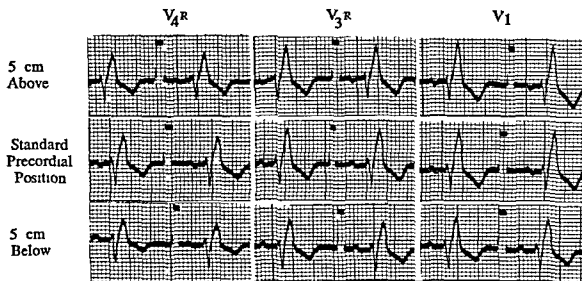
Sinus bradycardia is present because the cardiac rate is below 60 beats per minute and the pace maker is in the sinus node

A right complete bundle branch block is present because of the wide M-shaped QRS complexes (0.16 second) in leads  $V_1$ ,  $V_2$  and  $V_3$  and because of the wide S waves in leads  $V_4$ ,  $V_5$  and  $V_6$

It is to be noted that the first portions of the QRS complexes are essentially normal and represent a horizontal electrocardiographic position. For example, in lead  $aV_1$ , the first portions of the QRS complexes in this lead are qR waves which are similar to the first portions of the QRS complexes in lead  $V_6$ . In lead  $aV_F$ , the first portions of the QRS complexes are rS waves and they resemble the first portions of the QRS complexes of lead  $V_1$ .

**Case Summary**

A diagnosis of coronary arteriosclerosis with coronary insufficiency and angina pectoris was made. A 2:1 AV block associated with anginal pain developed after moderate exercise (see below). Teleoroentgenogram of the chest showed no evidence of cardiac enlargement. The blood pressure was 140/80. Special leads taken over the right chest show r waves followed by R prime waves of increased width which are characteristic of a right complete bundle branch block.

Lead  $V_4$  after exercise

I A

II C

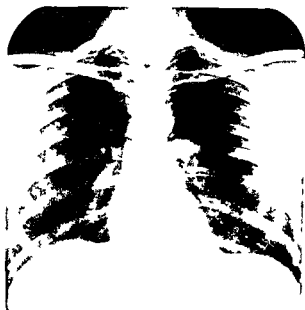
*Explanation*

The S T segments may be elevated normally 1.5 mm in lead  $V_2$  (see appendix). Large S T segment shifts are more common when the T waves are tall. The Q waves in leads II, III and  $aV_F$  are due probably to septal depolarization because of the clockwise rotation of the heart about the long axis which, electrically speaking, places the left side of the interventricular septum toward the left foot electrode.

The electrocardiographic position is semivertical. The ventricular complexes of lead  $aV_F$  resemble those of leads  $V_5$  and  $V_6$  and the QRS complexes of lead  $aV_L$  are small. Sinus rhythm, rate 70 beats per minute.

*Case Summary*

Intercostal neuritis. No heart disease. Heart normal size, shape and position. The cardiothoracic ratio is 0.37.



I C

II B

### Explanation

An incomplete AV block with dropped beats is present as certain P waves are not followed by QRS complexes indicating no atrioventricular conduction. The block is 2:1 in all leads except in lead aV<sub>F</sub> in which case the block is variable. At times it is difficult to differentiate second degree from third degree AV block. The differentiation is aided by exercising the patient. With complete AV block the atrial rate increases with exercise, however, the ventricular rate remains unchanged. With second degree AV block both atrial and ventricular rates increase with exercise. The tall P waves in leads II and III are consistent with chronic pulmonary disease. Pulmonary emphysema is a common cause of this finding.

### Case Summary

Pulmonary emphysema and coronary arteriosclerosis. The vital capacity is 50 per cent of normal and 70 per cent of the air is expired in three seconds. Maximum breathing capacity is 40 per cent of normal. Teleoroentgenogram shows low diaphragms and horizontal ribs. The heart is not enlarged. The cardiothoracic ratio is 0.43. There is calcification of the aorta. The teleoroentgenogram is typical of pulmonary emphysema.



A

*Explanation*

Left complete bundle branch block is present. The widest QRS complexes of the three standard leads measure 0.13 second. Wide positive QRS complexes in leads  $V_5$  and  $V_6$  with evidence of atrial depolarization help make this diagnosis. The presence of P waves shows that the activation wave was transmitted from the atria and helps to rule out wide QRS complexes which originate from a ventricular pacemaker. The intrinsicoid deflections are prolonged in lead  $V_6$  measuring 0.09 second (upper limit of normal is 0.05 second see appendix). A digitalis effect is present. The Q-T intervals were corrected for the increased width of the QRS complexes to a normal width of 0.08 second see appendix. In lead  $V_6$  the Q-T intervals corrected in this way measure 0.28 second. The lower limit of normal for a cardiac rate of 94 beats per minute is 0.28 second. Thus the interval in this case is at the lower limit of normal and is therefore in keeping with a digitalis effect.

*Case Summary*

Angina pectoris probably secondary to coronary arteriosclerosis. Heart normal size, shape and position. The aorta is prominent. Slight calcification of the aorta arch is present. The cardiothoracic ratio is 0.41.



### Answer

I B

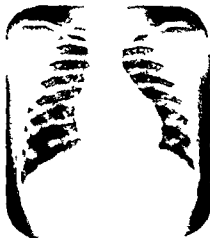
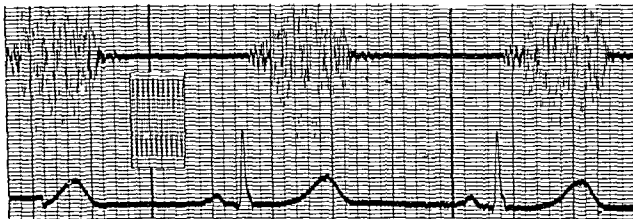
II D

### Explanation

Right ventricular hypertrophy is present as the R waves in lead  $V_1$  are tall measuring 13.0 mm (upper limit of normal is 9.0 mm for this age see appendix) Note the low standardization in the precordial leads Sinus arrhythmia is present because the R-R intervals vary with respiration

### Case Summary

Infundibular pulmonic stenosis without atrial or ventricular septal defects diagnosed by cardiac catheterization Phonocardiogram showed a diamond shaped systolic murmur recorded from the pulmonary area This is typical of pulmonary stenosis The heart is enlarged slightly in its transverse diameter The lung fields are clear The pulmonary vascular markings are decreased



## Answer

A

## Explanation

The tracing fits the criteria of an incomplete right bundle branch block because of the rsR prime waves and the delay of the onset of the intrinsicoid deflections to 0.06 second in lead V<sub>1</sub>. An incomplete right bundle branch block usually delays the onset of the intrinsicoid deflection so that it falls between 0.05 and 0.075 second. A right ventricular diastolic overload commonly produces tracings of this sort. The tracing also shows a digitalis effect which is clearly revealed in lead aV<sub>F</sub> where there is sagging of the S-T segments. The T waves are of the minus plus type and the terminal portions of the T waves are small in this lead. The Q-T intervals tend to be short.

## Case Summary

Atrial septal defect is present. At surgery the pulmonary artery pressure was elevated. The atrial defect was an ostium secundum 10 mm in diameter which was closed readily. Two right pulmonary veins drained into the superior vena cava and one drained into the right atrium. X-ray taken eight days post-operatively shows increased markings in both lung bases. The apex of the cardiac shadow is displaced to the left.





I B

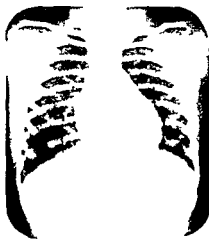
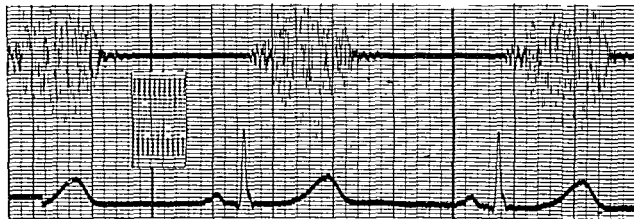
II D

### *Explanation*

Right ventricular hypertrophy is present as the R waves in lead  $V_1$  are tall measuring 13.0 mm (upper limit of normal is 9.0 mm for this age see appendix) Note the low standardization in the precordial leads Sinus arrhythmia is present because the R-R intervals vary with respiration

### *Case Summary*

Infundibular pulmonic stenosis without atrial or ventricular septal defects diagnosed by cardiac catheterization Phonocardiogram showed a diamond shaped systolic murmur recorded from the pulmonary area This is typical of pulmonary stenosis The heart is enlarged slightly in its transverse diameter The lung fields are clear The pulmonary vascular markings are decreased



## Answers

I A

II B

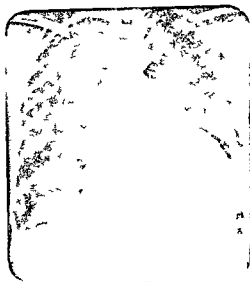
### Explanation

Atrial fibrillation is present because of the absence of clearly distinguishable P waves and because of the irregular R-R intervals. Left ventricular hypertrophy is present because of the deep S waves in lead  $V_2$  and fairly tall R waves in lead  $V_3$ .

The U waves are abnormally large especially in leads  $V_2$  and  $V_3$ . The U waves represent the after potential and the period of greatest excitability of the ventricle. They tend to be of greatest magnitude in lead  $V_3$  and usually they point in the same direction as the T waves. The waves are unusually large with ventricular hypertrophy, bradycardia, complete heart block, hyperthyroidism, hyperkalemia and hypokalemia, digitalis, epinephrine and hypercalcemia. In certain electrolyte disturbances the U waves are inverted.

### Case Summary

Rheumatic mitral insufficiency and stenosis of long duration. Clinically the patient has a grade III systolic and grade II early diastolic murmur at the mitral area. The heart is enlarged markedly to the left. There is fullness in the region of the pulmonary conus. The aortic knob is small. The lung fields are congested.



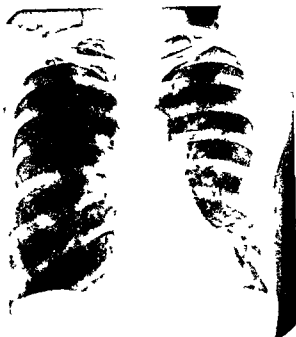
C

*Explanation*

Right atrial enlargement is suggested by the tall peaked P waves in leads II and III. It should be noted that the heart is relatively low in the chest in relation to the rib cage so that the electrodes were placed high with respect to the heart. The fourth intercostal space anteriorly passes across the base of the heart. In normal individuals the electrodes are placed over the anterior surface of the heart. In this patient the high position of the electrodes helps to explain the relatively small r and deep S waves in lead  $V_5$  and  $V_6$ . The tall R waves in lead  $V_1$  and deep S waves in lead  $V_6$  suggest right ventricular hypertrophy.

*Case Summary*

Advanced pulmonary emphysema. Hematocrit 56 per cent. The teleoroentgenogram is typical of pulmonary emphysema. The ribs are horizontal. There is decreased density at the right upper half of the lung. Heart is not definitely enlarged. The cardiothoracic ratio is 0.44.



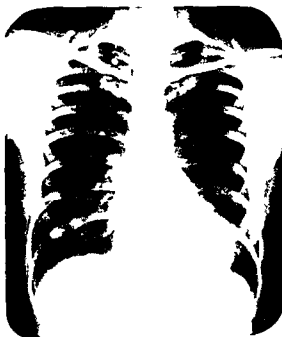
C

*Explanation*

Atrial enlargement is present because of the tall peaked P waves in leads II III and aV<sub>F</sub>. In lead II the P amplitudes exceed the upper limit of normal of 2.5 mm measuring 3.0 mm (see appendix). The differentiation between right and left atrial enlargement often is uncertain especially if determined from the limb leads. Left ventricular hypertrophy is indicated by the high R waves in lead V<sub>6</sub> which measure 38.0 mm (upper limit of normal is 22.0 mm for adults see appendix). The sum of the amplitudes of RV<sub>5</sub> plus SV<sub>1</sub> exceeds the upper limit of normal of 35.0 being 47.0 mm. A digitalis effect is indicated by the short Q-T intervals which in lead V<sub>2</sub> are 13 per cent below average. Sinus tachycardia is present with a rate of 110 beats per minute.

*Case Summary*

Severe bronchial asthma thirty years. Essential hypertension twenty years. Patient is taking digitalis leaf 0.2 gm (3 grains) daily. Teleoroentgenogram shows a heart which is enlarged to the left. The cardiothoracic ratio is 0.57.



A

*Explanation*

Right ventricular hypertrophy is present. The R waves are tall in lead  $V_1$  and exceed the upper limit of normal of 7.0 mm. They measure 11.0 mm. The intrisicoid deflections are delayed in lead  $V_1$  being 0.05 second. A digitalis effect shows clearly in lead  $V_3$  in which the S-T segments are saucer shaped and the Q-T intervals are at the lower limit of normal. They measure 0.28 second for a cardiac rate of 93 beats per minute (lower limit of normal is 0.28 second see appendix)

*Case Summary*

Pulmonary emphysema advanced. Resting arterial oxygen saturation 80 per cent. After exercise 65 per cent. At necropsy the heart weighed 420 grams. Marked dilatation of the right atrium and the right ventricle. The left ventricle measured 10 to 15 mm. and the right ventricle 3 to 10 mm. in thickness. Teleoroentgenogram shows the heart enlarged markedly and lifted from the diaphragm. This suggests right ventricular hypertrophy. The ribs are horizontal. There is an increased density at the right lung base which is in keeping with a diagnosis of bronchiectasis and pulmonary fibrosis which was present at necropsy. The cardiothoracic ratio is 0.61.



I A

II B

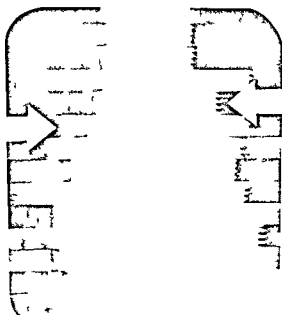
### Explanation

The electric axis of QRS is deviated to the right as it is 116 degrees (upper limit of normal for a small series of normals is 102 degrees see appendix) The axis was obtained by subtracting the amplitudes of the positive and negative waves in lead I which equals minus 4 mm and performing the same calculation in lead III which gives plus 9 mm The axis was found by using these figures in the table in the appendix

The predominantly positive QRS complexes and R prime waves in lead  $V_1$  suggest an incomplete right bundle branch block

### Case Summary

Atrial septal defect revealed by cardiac catheterization A phonocardiogram taken from the pulmonary area revealed a systolic and early diastolic murmur The heart is enlarged in its transverse diameter The cardiothoracic ratio is 0.50 The pulmonary conus area is enlarged and the vascular markings at the inner lung fields are increased A six foot X ray kymogram shows increased pulsations of the pulmonary arteries (see below)



Enlarged photograph of kymogram

I D

II A

*Explanation*

Supraventricular tachycardia is present with a cardiac rate of 136 beats per minute. It is difficult in tracing this to determine if this is sinus or atrial tachycardia. With sinus tachycardia the cardiac rate is usually less than 160 beats per minute. Sinus arrhythmia is present or appears on deep breathing; the cardiac rate slows gradually with carotid sinus pressure and the P waves are usually clearly distinguishable. With atrial tachycardia the cardiac rate is usually but not always greater than 160 beats per minute. Sinus arrhythmia is not present and does not appear on deep breathing. Carotid sinus pressure either produces sinus rhythm abruptly or has no effect and the P waves are usually buried in the T waves.

Left ventricular hypertrophy is diagnosed because of the high voltage of the QRS complexes with discordant J points, ST segments and T waves in leads  $V_4$ ,  $V_5$  and  $V_6$  along with the deep S waves in leads  $V_1$  and  $V_2$ . The relatively large Q waves in leads  $V_5$  and  $V_6$  probably are due to septal hypertrophy.

*Case Summary*

Three years ago the patient had an anterior myocardial infarct. This is revealed in the tracing by the low R waves in lead  $V_3$ . This usually indicates anterior infarction especially when the R waves in lead  $V_3$  are lower than the R waves in lead  $V_2$ . Small r waves in lead  $V_3$  occur in a few individuals with left ventricular hypertrophy. The tracing also shows a digitalis effect.

## Answer

C

## Explanation

The tracing is abnormal as the T waves in leads aV<sub>F</sub>, II and V<sub>4</sub> through V<sub>6</sub> are low relative to the height of the R waves. Also the ST segments are slightly negative in these leads. These changes are consistent with an abnormality of the myocardium and could be secondary to longstanding severe anemia (primary T wave changes), however, left ventricular hypertrophy also often produces similar changes (secondary T wave changes). The tracing does not indicate coronary artery disease as electrolyte disturbances, drugs, and certain diseases which effect the myocardium such as rheumatic fever, can produce similar abnormalities. The tracing is not diagnostic of hypertensive heart disease as this cannot be diagnosed from the electrocardiogram. Left ventricular enlargement which may be associated with hypertensive heart disease can be diagnosed from the tracing, but it can be produced also by aortic stenosis or insufficiency or other disease states. Normal sinus rhythm. Rate 76. Semivertical electric position.

## Case Summary

Sickle cell anemia. Hemoglobin 5.0 grams. Blood pressure 110/68. The heart is enlarged to the left. The cardiothoracic ratio is 0.56. No congestive heart failure. Blood electrolytes were normal.





## Answers

I D

II A

### Explanation

Supraventricular tachycardia is present with a cardiac rate of 136 beats per minute. It is difficult in tracing this to determine if this is sinus or atrial tachycardia. With sinus tachycardia the cardiac rate is usually less than 160 beats per minute, sinus arrhythmia is present or appears on deep breathing, the cardiac rate slows gradually with carotid sinus pressure, and the P waves are usually clearly distinguishable. With atrial tachycardia the cardiac rate is usually but not always greater than 160 beats per minute, sinus arrhythmia is not present and does not appear on deep breathing, carotid sinus pressure either produces sinus rhythm abruptly or has no effect, and the P waves are usually buried in the T waves.

Left ventricular hypertrophy is diagnosed because of the high voltage of the QRS complexes with discordant J points, ST segments, and T waves in leads  $V_4$ ,  $V_5$ , and  $V_6$ , along with the deep S waves in leads  $V_1$  and  $V_2$ . The relatively large Q waves in leads  $V_5$  and  $V_6$  probably are due to septal hypertrophy.

### Case Summary

Three years ago the patient had an anterior myocardial infarct. This is revealed in the tracing by the low R waves in lead  $V_3$ . This usually indicates anterior infarction, especially when the R waves in lead  $V_3$  are lower than the R waves in lead  $V_2$ . Small r waves in lead  $V_3$  occur in a few individuals with left ventricular hypertrophy. The tracing also shows a digitalis effect.

I A

II A

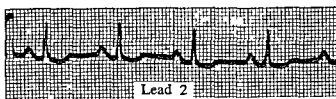
*Explanation*

Atrial flutter is diagnosed because of the lack of isoelectric periods in leads  $aV_R$  and  $aV_F$ . Isoelectric periods are present however in leads  $V_1$  and  $V_2$ . The relatively low atrial flutter waves in lead I with taller flutter waves in leads II and III suggest that the atrial depolarization pathway is almost perpendicular to a line connecting the lead I electrodes.

Right ventricular hypertrophy is suggested by the qr waves in lead  $V_1$  and right axis deviation of the QRS complexes.

*Case Summary*

The patient has rheumatic mitral stenosis and had a mitral commissurotomy. The mitral orifice was 0.5 cm. Atrial fibrillation was present on 12/26/53 and a sinus rhythm was present on 6/13/55 (see below). Teleoroentgenogram of the heart shows fullness in the region of the pulmonary conus. The walking point (junction of left atrial appendage and the left ventricle as seen in the P-A view) is displaced toward the apex. The aortic knob is small. The hilar markings are increased. The cardiothoracic ratio is 0.58.



June 13 1955



### Answers

I A

II A

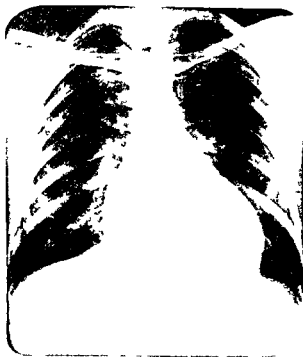
### Explanation

The time of onset of the intrinsicoid deflection in lead  $V_6$  is delayed. The initial portions of the QRS complexes in this lead are isoelectric which is revealed by comparing lead  $V_6$  against lead  $V_2$  in the simultaneous recording.

A myocardial infarct is suggested by the Q waves in leads I and  $aV_L$  and low R waves in leads  $V_3$ . The Q/R ratio in lead  $aV_L$  usually does not exceed 0.75 (see appendix). In this tracing it measures 1.5. The shape of the QRS-T waves in leads  $V_5$  and  $V_6$  suggest left ventricular hypertrophy; however, the voltage of the R waves is unusually low for this condition. This may be due to the myocardial infarct or to edema or to other causes which reduce voltage.

### Case Summary

Clinically the patient had a myocardial infarct three years ago and has had three attacks of pulmonary edema since. The heart is enlarged to the left. There is rounding at the apex. The cardiothoracic ratio is 0.55. Cardiac dilatation is probable.



B

*Explanation*

The P R intervals are short and in lead II measure 0.10 second. The QRS intervals are long (0.11 second). The P R intervals are especially short when considered in the light of the slow cardiac rate. Slurring on the upstroke of the R waves in leads I and  $V_2$  through  $V_6$  is characteristic of this condition. This suggests an anomalous atrioventricular conduction. Normal sinus rhythm rate 71 beats per minute.

*Case Summary*

There is no definite evidence of cardiac disease. The patient has a Wolff Parkinson White syndrome and has attacks of tachycardia which lasts from ten minutes to hours. These have been present for years. The patient has angina pectoris on effort. Radiologically the heart is not enlarged in its transverse diameter. The aortic knob is large and prominent.



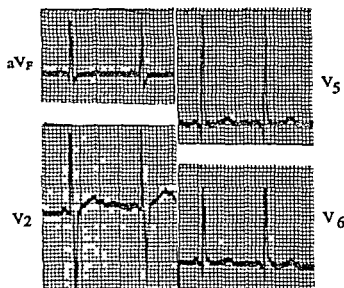
B

*Explanation*

The tracing is suggestive of left and right ventricular hypertrophy. The tall QRS complexes with fairly deep Q waves and relatively low T waves in leads  $V_5$  and  $V_6$  suggest left ventricular hypertrophy. Hypertrophy of the interventricular septum frequently produces large Q waves in the leads overlying the left ventricle. The tall R waves in leads  $V_1$  and  $V_2$  suggest right ventricular hypertrophy. One cannot rule out the possibility of myocarditis or pericarditis as a cause for the T wave changes in leads  $aV_F$ ,  $V_2$ ,  $V_5$  and  $V_6$  and ST segment shifts in leads II, III,  $aV_F$ ,  $V_5$  and  $V_6$ . As the ST segments are concordant with the tall R waves in leads  $aV_F$  and  $V_5$ , it is probable that myocardial injury is present in addition to ventricular hypertrophy. A tracing taken six months previously showed more normal ST segments and T waves in these leads (see below). Serial tracings of this sort are important when trying to differentiate ST-T segment and T wave changes of hypertrophy from those due to other factors such as myocarditis, pericarditis or electrolyte disturbances.

*Case Summary*

Rheumatic mitral stenosis and insufficiency. The child has been in bed for one year with typical rheumatic fever. There was no definite evidence of pericarditis. Auscultation of the heart revealed a grade II systolic murmur and an early diastolic murmur at the mitral area. Teleoroentgenogram showed fullness in the region of the pulmonary conus with increased vascular markings.



Tracing Taken  
Six Months Ago



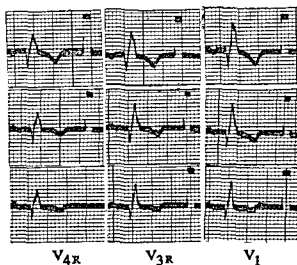
A

*Explanation*

The duration of the QRS complexes measured in lead II is 0.13 second and the time of onset of the intrinsicoid deflection in lead  $V_1$  is 0.09 second. A right complete bundle branch block is diagnosed when the durations of the QRS complexes are 0.12 second or greater, rsR' prime waves are present in lead  $V_1$  and the time of onset of the intrinsicoid deflection (R' prime wave) is delayed. Usually the time of onset in lead  $V_1$  is 0.08 second or greater. Additional leads taken over the right chest show in certain leads small initial r waves and R' prime waves which are characteristic of a right bundle branch block.

*Case Summary*

Essential hypertension for ten years. Mild angina pectoris. Patient had a thoracolumbar sympathectomy two years ago. The heart is not enlarged. There is slight widening of the great vessel shadow. The cardiothoracic ratio is 0.36. The tracing is typical of a right complete bundle branch block without other cardiac abnormalities, e.g. without myocardial ischemia, injury or infarction.

5 cm  
AboveStandard  
recording  
positions5 cm  
Below

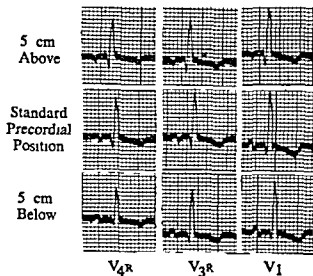
C

*Explanation*

The tracing fits the criteria for right ventricular hypertrophy. Some of these are qR waves in leads  $V_{3R}$  or  $V_1$ , R waves in lead  $V_1$  which are greater than 7.0 mm, an R/S ratio in lead  $V_1$  greater than 1.0 and a delay in the onset of the intrinsicoid deflection from 0.035 to 0.05 second.

*Case Summary*

Primary pulmonary hypertension. Cardiac catheterization showed a pressure in the pulmonary artery of 83/41 mm of mercury, in the right ventricle 83/2 and in the right atrium 8/1. Teleoroentgenogram of the chest showed enlargement in the region of the pulmonary conus and right pulmonary artery. The heart appears to be shifted from the diaphragm indicating right ventricular hypertrophy. The cardiothoracic ratio is 0.49. X-ray kymogram showed right ventricular and pulmonary arterial pulsations which exceeded those of the left ventricle and aorta.



B

*Explanation*

Hypokalemia is suggested by the presence of the large U waves which are superimposed on the downstroke of the T waves. The sagging of the S-T segments in leads  $V_4$ ,  $V_5$  and  $V_6$  is consistent with this diagnosis. The tracing before treatment was taken when the patient's serum potassium was 3.2 mEq per liter which is 64 per cent of the normal. The time measured from the beginning of the Q waves to the peak of the T waves was prolonged as compared with records made after treatment. The patient was treated with intravenous potassium chloride after which the serum potassium was 4.9 mEq per liter which is normal. As a result of treatment the U waves became smaller, the time from the beginning of the QRS complexes to the peak of the T waves shortened and the S-T segment shifts became more normal.

*Case Summary*

Intestinal obstruction due to neoplasm. The hyperkalemia was due to the potassium loss from the stomach following gastric suction for three days. X-ray of the chest showed that the heart was normal size, shape and position. The lungs were clear.



## A and C

*Explanation*

An incomplete AV block is present because the P R intervals are prolonged. The intervals in lead II measure 0.32 second; the upper limit of normal is 0.20 second for rates above 70 beats per minute. Also, right bundle branch block is present because of the wide QRS complexes (0.13 second) which are predominantly positive in lead V<sub>1</sub>. Wide S waves are present in lead V<sub>6</sub>. In addition, the tracing is abnormal because of negative S T segment shifts in leads I, V<sub>3</sub>, and V<sub>4</sub> and positive shifts in lead III. These suggest at least injury to the posterior aspect of the left ventricle. This is confirmed by comparison with a tracing taken two weeks previously when the patient was receiving the same amount of digitalis but before chest pain had developed and in which the S T segments were more isoelectric (see below). Characteristically when S T segment shifts occur secondary to a right bundle branch block, they are discordant with the wide terminal portions of the QRS complexes in most leads. In this case they are concordant and therefore suggest the presence of cardiac disease in addition to the block in the right bundle of His.

*Case Summary*

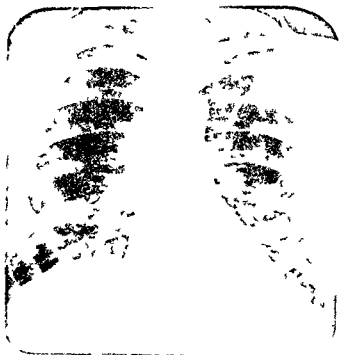
Arteriosclerotic coronary artery disease with a possible posterior myocardial infarct. An infarct was suspected because the S T segment shifts (injury shifts) persisted for more than 24 hours. Average blood pressure 180/72. Radiologically the transverse diameter of the heart is at the upper limit of normal.



One day later a 3:2 AV block with Wenckebach periods appeared. Lead V<sub>1</sub>



Tracing 2 weeks previously



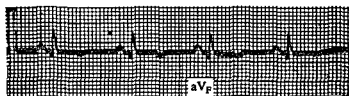
B

*Explanation*

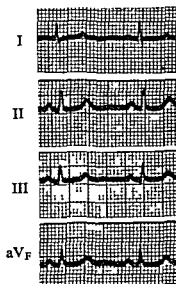
The tracing is suggestive of a posterior myocardial infarct. The Q waves in lead  $aV_F$  measure 0.03 second and are not wide enough to be typical of a posterior myocardial infarct (they should measure 0.04 second in duration or more). The Q/R ratios in lead  $aV_F$  do not indicate an infarct as the ratios are only 0.17 (they should exceed 0.28 to be indicative of an infarct). The Q waves in lead  $aV_F$  do not disappear with inspiration which is in keeping with a posterior infarct. The tracing taken before the infarct showed no Q waves in lead  $aV_F$  (see below). After comparing these two tracings a diagnosis of posterior myocardial infarction can be made with greater assurance.

*Case Summary*

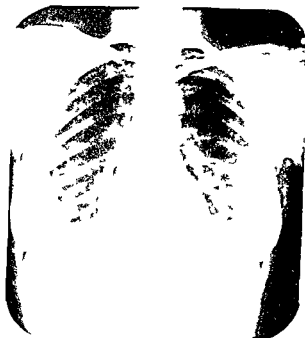
Old posterior myocardial infarct. The patient had severe chest pain of long duration with an increased sedimentation rate and white count. During the pain the T waves were inverted in lead  $aV_F$  (see below). Heart normal size, shape and position. The cardiothoracic ratio is 0.39.



Tracing During Chest Pain



Before the Infarct



B

*Explanation*

The positive S T segment shifts in leads I II aV<sub>L</sub> V<sub>4</sub> V<sub>5</sub> and V<sub>6</sub> indicate recent injury to the subepicardial surface of the left ventricle. This may be due to injury from various causes such as trauma surgery pericardial infection etc with involvement of the subepicardium.

*Case Summary*

Congenital bilateral polycystic kidneys Blood pressure 230/120 Death in uremia Postmortem examination showed advanced fibrinous pericarditis. The injury shifts of the electrocardiogram developed at the time a friction rub appeared a few days before death. Teleoroentgenogram showed the heart was enlarged to the left.



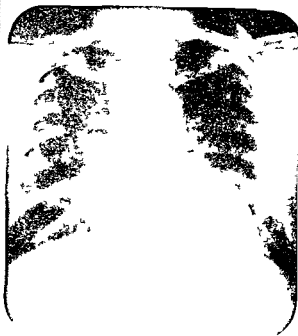
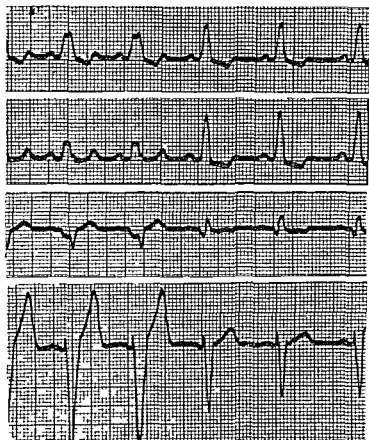
B

*Explanation*

An incomplete left bundle branch block is present. The QRS complexes are wide measuring 0.12 second and the time of onset of the intrinsicoid deflection in lead  $V_6$  is delayed being 0.08 second (upper limit of normal is 0.05 second see appendix). The QRS complexes are predominantly positive in the leads overlying the left ventricle (leads  $V_5$  and  $V_6$ ) and there is evidence of atrial activity (P waves). The normal relationship between P waves and QRS complexes suggests that normal AV conduction has taken place and helps to distinguish left bundle branch block from ventricular ectopic beats or ventricular tachycardia. The incomplete block was paroxysmal alternating with periods of complete block (see below). A digitalis effect is present. Sinus rhythm rate 88 beats per minute.

*Case Summary*

Arteriosclerotic coronary artery disease. Five years ago the patient had severe chest pain which was clinically a myocardial infarct. Angina pectoris since. Highest blood pressure 168/100 and lowest pressure 140/80. Radiologically the heart is enlarged to the left and there is rounding at the apex. There is slight congestion of the lung fields. The cardiothoracic ratio is 0.53.



Change from complete left bundle branch block to incomplete left bundle branch block

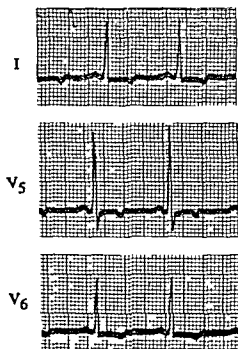
D

*Explanation*

The T waves in leads aVL, V<sub>5</sub> and V<sub>6</sub> are abnormal as they are negative in the presence of clearly positive QRS complexes. The terminal coving of these waves suggests myocardial ischemia. A tracing taken three months previously showed very little change in the T waves indicating that the ischemia was chronic (see below). The tall R waves in lead I and deep S waves in lead V<sub>2</sub> suggest left ventricular hypertrophy. It is difficult often to differentiate myocardial ischemia from left ventricular hypertrophy. In the former terminal coving of the S-T segments is characteristically found and the amplitudes of the QRS complexes are not unusually tall. In the latter the S-T segments are straight generally and the amplitudes of the R waves are usually tall. A transmural anterior myocardial infarct is not present as large Q waves or QS waves do not occur in the pre-cordial leads. Electrolyte disturbances do not produce terminal coving of the S-T segments.

*Case Summary*

Diabetes mellitus Essential hypertension Highest blood pressure 256/120 Lowest pressure 185/105 Coronary arteriosclerosis with coronary insufficiency and myocardial ischemia No definite cardiac enlargement The cardiothoracic ratio is 0.46 Grade III Keith Wagener retinopathy



Three months previously



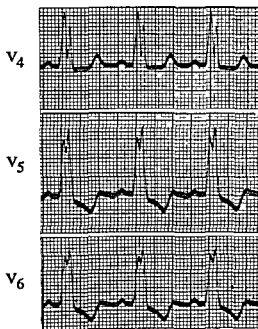
B

*Explanation*

The QRS complexes are wide measuring 0.16 second in lead I (upper limit of normal is 0.10 second see appendix). The QRS complexes are predominantly positive in the leads from the left chest. M-shaped QRS complexes are seen in lead  $V_4$ . There is evidence of atrial activity as shown by the P waves which precede the QRS complexes. Ventricular ectopic beats are present as they occur early in time, are wide, exceeding 0.10 second, are followed by secondary T waves and are not preceded by P waves. The ventricular premature beats are followed by full compensatory pauses (the sum of the R-R distance immediately in front of the ectopic beat and the R-R distance immediately in back of the ectopic beat is equal to the sum of two complete heart cycles in which no ectopic beat occurs). This means that the rhythm of the sinus node is not altered by ventricular ectopic beats because retrograde conduction from ventricles to atria does not occur in this case. The sagging of the S-T segments in leads  $V_4$ ,  $V_5$  and  $V_6$  and other leads is slightly suggestive of a digitalis effect. The tracing below shows leads  $V_4$ ,  $V_5$  and  $V_6$  taken before digitalis was administered. In this tracing the S-T segments are convex upward and the Q-T intervals are longer.

*Case Summary*

Arteriosclerotic coronary artery disease with coronary insufficiency and angina pectoris. Paroxysmal nocturnal dyspnea. Left ventricular congestive heart failure, severe. Radiologically, the heart is rounded at the apex. The cardiothoracic ratio is 0.52, indicating an increased transverse cardiac diameter. The great vessel shadow is enlarged. Calcification is present in the aortic knob. There is moderate congestion of both lungs.



Before digitalis



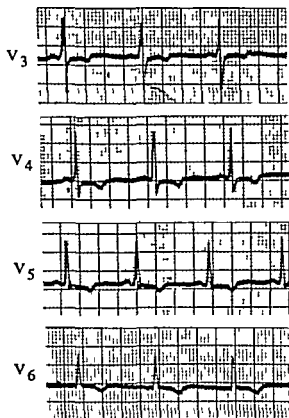
E

*Explanation*

The P R intervals are short and the P waves are inverted slightly in lead aV<sub>F</sub> and in most of the precordial leads. The cardiac pacemaker is in the AV node and there is retrograde conduction from the AV node through the atria toward the sinus node. The pacemaker is low in the AV node. Thus the atria and ventricles are activated almost simultaneously. Negative S T segment shifts in leads V<sub>5</sub> V<sub>6</sub> and other leads are consistent with coronary arteriosclerosis with insufficiency or digitalis or both. A tracing taken after chest pain but before digitalis is shown below.

*Case Summary*

Hypertensive arteriosclerotic coronary artery disease with severe angina pectoris. X ray showed that the heart was at the upper limit of normal in the transverse diameter. Average blood pressure 158/100.



Tracing taken three days after severe chest pain



*Answer*

B

*Explanation*

The low voltage of the QRS complexes in leads  $V_5$  and  $V_6$  was due to the presence of air between the heart and chest wall. A tracing taken after the lung had expanded showed taller R waves in the precordial leads recorded from the left side of the chest.

*Case Summary*

Left pneumothorax due to pulmonary tuberculosis. Heart normal size, shape and position. The cardiothoracic ratio is 0.41.





### Answer

I C

II B

### Explanation

Pressure on the carotid sinus was made during the recording of each lead. A long period of sinus arrest shows clearly in lead I and other leads. Sinus arrest is a temporary standstill of atria due often to increased vagal tone. The tracing is suggestive of an old posterior myocardial infarct because of the low R waves and inverted T waves in lead V<sub>6</sub>. Normally the R waves in this lead should exceed 4.0 mm and the T waves should not be more negative than 0.5 mm (see appendix). A ventricular escape beat is present in lead V<sub>3</sub>. The low voltage of the QRS complexes in the standard leads is in keeping with the diagnosis of myocardial infarction.

### Case Summary

Posterior myocardial infarction one year ago. Hypersensitive carotid sinus. The heart is rounded at the apex and it is enlarged in its transverse diameter. The cardiothoracic ratio is 0.51. The great vessel shadow is enlarged. There is calcification of the aorta.



## Answers

I B

II B

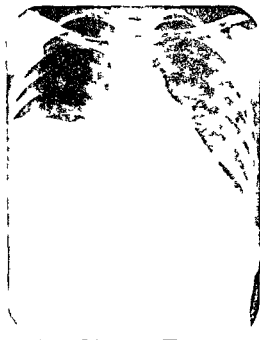
## Explanation

Regular vibrations occurring 60 times a second are present in most of the limb leads. The standardization is 5.0 mm in leads I and II and 8 mm in most of the other leads.

Atrial fibrillation is present because of the absence of demonstrable P waves along with the irregularity of the R-R intervals between the narrow QRS complexes which are conducted from the atria. Multifocal ventricular ectopic beats are present because the QRS complexes are of different form in certain leads indicating that these beats arise from different foci in the ventricles. A digitalis effect is present as shown by the sagging of the S-T segments. Digitalis intoxication could account for most of these findings. Low voltage of the QRS complexes is present in the standard leads.

## Case Summary

Coronary arteriosclerosis, coronary insufficiency, angina pectoris, and eventration of the right diaphragm. Teleoroentgenogram of the chest shows elevation of the diaphragm on the right. The cardiac size is difficult to determine in the presence of the diaphragmatic abnormality.



## Answer

A

## Explanation

An old posterior myocardial infarct is present. In lead  $aV_F$  the Q waves are wide and measure 0.05 second in duration. A posterior infarct is suggested when the Q waves measure 0.04 second or greater in this lead. The Q waves and inverted T waves in leads II and III support this diagnosis. Posterior peri infarction block is suggested by the presence of widening of the QRS complexes which measure 0.11 second in lead II with slurring of the terminal portions of the QRS complexes which is evident especially in lead  $aV_F$ .

## Case Summary

Posterior myocardial infarct eight months prior to this tracing. Angina pectoris. The heart is rounded at the apex. The cardiothoracic ratio is 0.48.



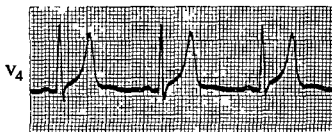
C

*Explanation*

A posterolateral infarct is suggested by the low R waves (3.0 mm) in lead  $V_6$  (lower limit of normal 4.0 mm see appendix). Posterior myocardial ischemia is suggested by the inverted T waves in leads II, III,  $aVF$  and  $V_6$ . In addition, the R waves in lead  $V_1$  are at the upper limit of normal being 7.0 mm. The R/S ratios in this lead are abnormal being 1.75 (upper limit of normal 1.0 see appendix). The electrocardiographic position of the heart is horizontal. Sinus bradycardia rate 58 beats per minute. A tracing taken a month prior to this showed no significant differences. The T waves in the right precordial leads at that time were relatively tall indicating that the ischemic process is static (see below).

*Case Summary*

Old myocardial infarct due to arteriosclerotic coronary artery disease. Heart is normal size, shape and position. The cardiothoracic ratio is 0.45.



Tracing taken one month previously



## Answer

C

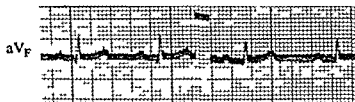
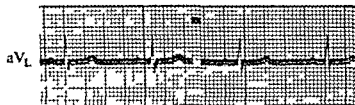
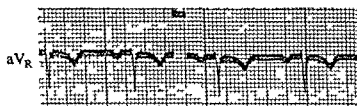
1

## Explanation

The large Q waves in lead  $aV_F$  and abnormal Q/R ratios indicate a posterior infarct. Ventricular ectopic beats are present in the limb leads. An intraventricular block is present as shown by the wide S waves in lead  $aV_L$  and wide R waves in lead  $aV_F$ . Lead  $aV_F$  taken with the paper speed running 75 mm per second shows slurring on the downstroke of the R waves which is further evidence of the intraventricular block. The unipolar limb leads recorded before the infarct show QRS complexes of normal duration. Widening of the terminal portions of the QRS complexes in lead  $aV_L$  and  $aV_F$  is not present (see below). Left ventricular hypertrophy is suggested by the R waves in lead  $V_4$  which are at the upper limit of normal.

## Case Summary

Posterior myocardial infarct. The heart is rounded at the apex. There is slight congestion of both lung fields. The cardiothoracic ratio is 0.42.



Before Infarct

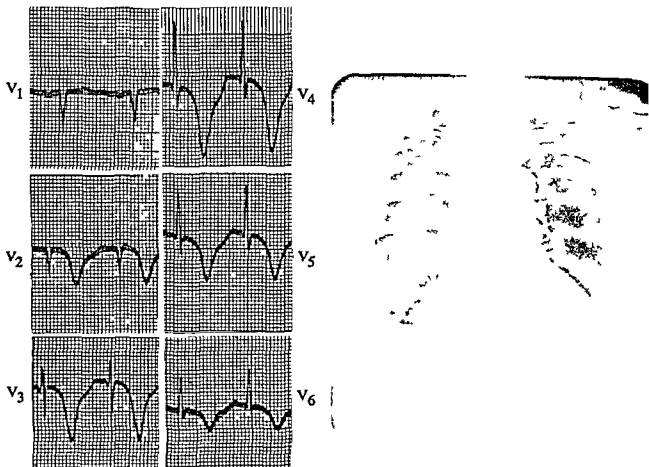


### Explanation

The QS waves in lead  $V_3$  are indicative of necrosis or death of the anteroapical aspect of the left ventricle. The T waves are of the coronary type with coving of the ST segments and with prolongation of the QT intervals. These findings suggest myocardial ischemia of the anteroapical, lateral and posterolateral aspects of the left ventricle. Sinus rhythm, rate 62 beats per minute. The tracing shown below was recorded four months ago at the onset of the precordial pain which was associated with the infarct. The coronary type T waves are prominent and QS waves are not present in lead  $V_3$ .

### Case Summary

Coronary arteriosclerosis with coronary occlusion and myocardial infarction four months ago. The heart is enlarged in its transverse diameter. The cardiothoracic ratio is 0.53. There is a fusiform aneurysm at the base of the ascending aorta. Calcification of the lymph nodes is present.



Tracing Taken Four Months Ago

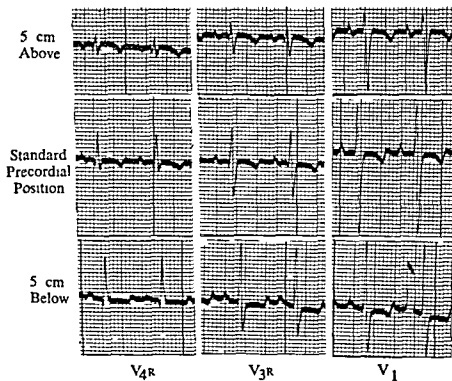
## C

*Explanation*

Right ventricular hypertrophy is suggested by the tall R waves in leads  $V_1$  and  $V_2$  which measure 11.0 and 23.0 mm respectively (they should not exceed 7.0 and 16.0 mm respectively see appendix) Also the time of onset of the intrinsicoid deflections in lead  $V_1$  is delayed slightly, measuring 0.04 second (upper limit of normal is 0.03 second see appendix) The additional leads to the right show the predominance of R waves which also indicate right ventricular hypertrophy. Left ventricular hypertrophy is suggested by the deep S waves in lead  $V_2$  which measure 34.0 mm (normally these waves should not be deeper than 29.0 mm see appendix) Sinus rhythm rate 83 beats per minute. Slight widening of the QRS complexes which measure 0.10 second (This is above the average which is 0.08 second see appendix)

*Case Summary*

Eisenmenger complex. Arterial oxygen saturation at rest 90 per cent which fell to 70 per cent after exercise. Arm to ear circulation time 7.5 seconds. Fluoroscopy showed increased pulsations of the pulmonary artery. The heart is enlarged in the region of the pulmonary conus and the apex is displaced downward and to the left. The cardiothoracic ratio is 0.56. The pulmonary arteries are enlarged especially on the right.



B

*Explanation*

Right complete bundle branch block of an unusual variety is present. The large P waves in lead II and lead  $V_1$  suggest right atrial enlargement. The enlargement involves the right atrium because in these leads the P waves are relatively narrow and tall and show no significant notching. Note that the right axis deviation of P waves which is typical of chronic cor pulmonale is not present. The P-R intervals are at the upper limit of normal. These findings are consistent with a diagnosis of Ebstein's syndrome. Anatomically this syndrome is characterized by right atrial enlargement with downward displacement of the tricuspid valve into a small right ventricle. Electrocardiographically tall peaked P waves in lead II and prolonged P-R intervals resulting from right enlargement are seen. The P axis is often about 40 degrees. Also a right complete bundle branch block with QRS intervals greater than 0.12 second is characteristically found. Usually there is a tendency to low voltage of the QRS complexes in the limb leads and in leads  $V_1$ ,  $V_2$  and  $V_3$  because of the thin upper portion of the right ventricle. There is normal voltage of the R waves in leads  $V_4$ ,  $V_5$  and  $V_6$  because of the relatively normal left ventricle. The intrinsoid deflection in lead  $V_1$  often is longer than that in lead  $V_6$ .

*Case Summary*

Ebstein's syndrome was diagnosed with the aid of an angiocardigram which demonstrated the large right atrium and the small right ventricle with downward displacement of the tricuspid valve. Teleoroentgenogram shows a heart which is enlarged in its transverse diameter. The right cardiac border is prominent. The vascularity of the lung fields is diminished.





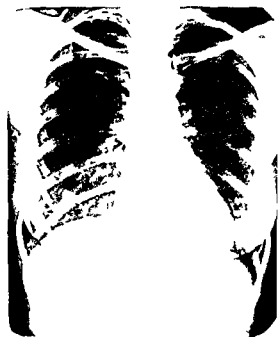
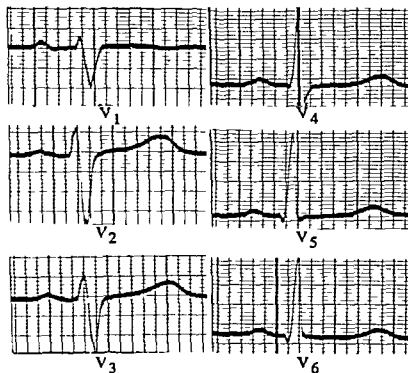
B

*Explanation*

The QS waves in lead  $V_3$  and small R waves in lead  $V_4$  are indicative of an anterolateral myocardial infarct. The infarct is subacute or chronic because the ST segments are essentially isoelectric and deep negative coved T waves are present in these leads. A tracing taken one year before the infarct is shown below. The r waves in lead  $V_1$  are unchanged. R waves were present in leads  $V_2$  and  $V_3$  before the infarct.

*Case Summary*

Typical myocardial infarct eight months previously. Heart enlarged slightly to the left and downward. The cardiothoracic ratio is 0.49.



Before the Infarct  
Paper Speed 75 mm Per Second

## Answers

I C

II E

## Explanation

Negative T waves in lead  $aV_L$  are found normally in vertically placed hearts. The QRS complexes and T waves have normal concordant relationships in this lead. The tracing is within normal limits. The electrocardiographic position is vertical because the ventricular complexes of lead  $aV_L$  resemble those of leads  $V_1$  and  $V_2$  and the ventricular complexes of lead  $aV_F$  resemble those of leads  $V_5$  and  $V_6$ .

## Case Summary

No heart disease. Normal male. Heart normal size, shape and position. The cardiothoracic ratio is 0.38.



B

*Explanation*

There is evidence of right ventricular and atrial hypertrophy. The former is shown in lead  $V_1$  by the tall R waves with discordant S T segments and T waves and the latter in lead  $V_1$  by the tall P waves which measure 2.5 mm (upper limit of normal 1.6 mm, see appendix). The clinical condition, pulmonary valvular stenosis, characteristically produces evidence of right ventricular hypertrophy in the electrocardiogram.

*Case Summary*

Congenital pulmonary valvular stenosis with atrial septal defect is present and was diagnosed by cardiac catheterization. Clinically there is a grade III systolic murmur and thrill over the pulmonary area. The intensity of the pulmonary second sound is diminished greatly. The heart is enlarged to the right and the apex is elevated. These findings suggest right atrial enlargement and right ventricular hypertrophy. The pulmonary arteries are enlarged which suggests poststenotic dilatation. The vascularity of the lung fields is diminished.



## C

*Explanation*

An incomplete right bundle branch block with ventricular hypertrophy is diagnosed because the requirements of an incomplete right bundle branch block are met and tall R prime waves are present in lead V<sub>1</sub> which measure 2.7 millivolts. Right ventricular hypertrophy in the presence of a right complete bundle branch block is diagnosed when the R prime waves in leads V<sub>1</sub> or V<sub>2</sub> measure more than 1.0 millivolt (Barker). Note that the standardization is lower than normal because of the tall QRS complexes which are present. A ventricular ectopic beat is present in lead V<sub>3</sub>.

*Case Summary*

Large ventricular septal defect diagnosed by oxygen data during cardiac catheterization. Oxygen saturation of blood from the right atrium was 67 per cent and from the right ventricle 77 per cent. Pulmonary and right ventricular hypertension. Pulmonary artery pressure 117/70 and right ventricular pressure 118/2. Findings compatible with Eisenmenger's complex or functional single ventricle. The heart is enlarged to the right and is elongated slightly to the left. The vascular markings are normal or increased.



A

*Explanation*

The R waves are tall in leads I and II being 13.0 and 17.0 mm respectively (upper limit of normal is 11.4 and 16.0 mm respectively for this age). The S waves in leads  $V_1$  and  $V_2$  are deep and the R waves in leads  $V_4$ ,  $V_5$  and  $V_6$  are relatively tall. The ST segments and T waves are relatively normal. Thus the diagnosis of left ventricular hypertrophy is made primarily on the basis of increased voltage of the QRS complexes. It is somewhat hazardous to make a diagnosis of left ventricular hypertrophy solely on the basis of increased voltage as the voltage may be increased by various causes such as a thin chest wall which places the precordial electrodes closer to the myocardium than is usual.

*Case Summary*

Coarctation of the aorta which was corrected surgically. Teleoroentgenogram of the chest shows the apex of the heart to be rounded due to left ventricular enlargement. The cardiothoracic ratio is 0.49. The lung fields are clear. Notching of the ribs is present in the region of the right lung base.



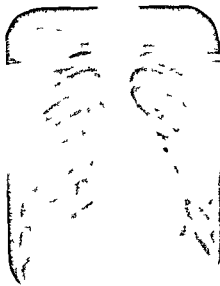
B

*Explanation*

In lead  $V_1$  small rSR prime waves are present with delay in onset of the intrinsicoid deflections of R prime to 0.05 second (in incomplete right bundle branch block it varies usually from 0.05 to 0.075 second) The S waves in lead  $V_6$  are of greater duration than the R waves Sinus rhythm rate 88 beats per minute

*Case Summary*

Congenital heart disease Atrial septal defect Cardiac catheterization showed a large interatrial left to right shunt which measured 2.6 liters per minute The oxygen saturation of blood from the superior vena cava was 61 per cent and from the right atrium was 81 per cent The right ventricular pressure was 32/0 Roentgenographically the heart is enlarged The pulmonary artery is prominent The aorta is small The vascularity of the lung fields is normal (typically it is increased)



B

*Explanation*

The infarct appears chronic. The QS waves in leads  $V_1$  and  $V_2$  and small  $r$  waves in leads  $V_3$  and  $V_4$  suggest a subendocardial infarct which is not transmural in that portion of the myocardium adjacent to these electrodes. In exceptional circumstances small  $r$  waves may be present in the presence of a transmural infarct if the infarct is small. In such cases the electrode picks up forces from the healthy myocardium obscuring the changes due to the infarct. The infarct in this tracing appears old (more than a few weeks) because the S-T segments are isoelectric. It is often difficult to determine the age of an infarct from the electrocardiogram as the presence or absence of segment shifts depends upon the acuteness of the onset of the infarct as well as on its age. Serial tracings are helpful in determining the age of the infarct because chronic infarcts show no significant changes while acute or subacute infarcts show progressive or regressive changes. Sinus bradycardia is present.

*Case Summary*

Typical myocardial infarct one year ago. The heart is at the upper limit of normal size. The cardiothoracic ratio is 0.44. Increased densities are present in the hilar regions.



I A

II A

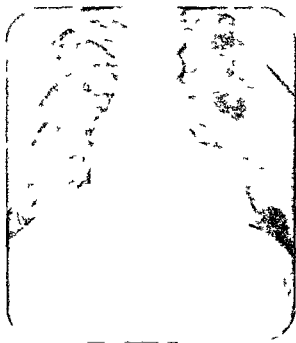
*Explanation*

The QS waves in leads  $V_2$ ,  $V_3$  and  $V_4$  and large Q waves in lead  $V_5$  indicate an anterolateral myocardial infarct. Q waves in lead  $V_5$  should not be deeper than 21 mm (see appendix). Normally the depth of the Q waves varies with the height of the associated R waves. The abnormally deep Q waves with respect to the height of the R waves in lead  $V_5$  is shown by an abnormal Q/R ratio (see appendix). With left ventricular hypertrophy the septal Q waves are usually less than 25 per cent of the height of the R waves. In this case they are greater.

The electrocardiographic position of the heart is horizontal because the ventricular complexes of lead  $aV_F$  resemble those of lead  $V_1$  and the ventricular complexes of lead  $aV_L$  resemble those of lead  $V_6$ . In the presence of cardiac disease the electric position usually does not correlate well with the anatomic position of the heart.

*Case Summary*

Hypertensive heart disease. Average blood pressure 168/98. Congestive heart failure. Myocardial infarct three years ago. Coronary arteriosclerosis and angina pectoris. The heart is enlarged markedly to the left. The cardiothoracic ratio is 0.55. X ray kymogram shows absent pulsations over the left lateral border of the heart.





E

*Explanation*

Saucer shaped S T segments with relatively short Q T intervals characteristic of digitalis are present. The intervals in lead  $V_4$  measure 0.32 second (lower limit of normal for a cycle length of 0.95 is 0.32 second see appendix). The magnitude of the J and S T segment shifts in lead  $V_4$  and other leads is greater than can be accounted for on the basis of therapeutic amounts of digitalis and suggests subendocardial injury as well. Large U waves are present in lead  $V_3$  and other leads.

*Case Summary*

Hypertensive heart disease. Blood pressure 160/100 for ten years. Mild left ventricular congestive heart failure. Angina pectoris on effort. The heart appears at the upper limit of normal size. The aortic knob is prominent. The lung fields are clear.



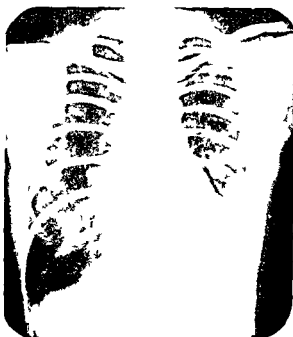
A

# Explanation

The tracing suggests abnormalities of the atria and right and left ventricles. Atrial fibrillation is present. The QR waves in lead  $V_1$  suggest right ventricular hypertrophy while left ventricular hypertrophy is suggested by the deep S waves in lead  $V_4$  and tall R waves with discordant J shifts. ST segments and T waves in lead  $V_6$ . A digitalis effect is present. The durations of the QRS complexes in lead  $V_1$  are less than those in lead  $V_2$  because the first portions of the QRS complexes in lead  $V_1$  are isoelectric and correspond in time to the writing of the r waves in lead  $V_2$ .

# Case Summary

Rheumatic mitral insufficiency and stenosis. There was an apical grade III systolic murmur and thrill and an apical grade II diastolic murmur. Heart enlarged markedly to the left. The pulmonary conus is large. The cardiothoracic ratio is 0.67.



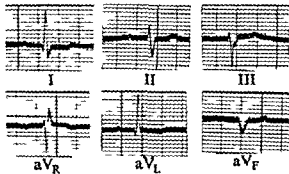
C

## Explanation

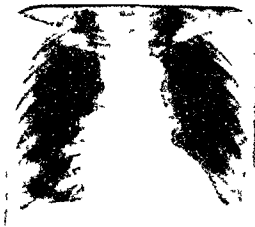
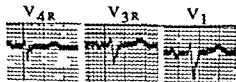
Lead I appears to be upside down. This could be due to switching of the lead I wires when applied to the patient's arms. The lead wire labeled LA then would be on the right arm of the patient and would be recording the potential variations from the right arm. The lead wire labeled RA would be on the left arm of the patient and would be recording the potential variations from the left arm. The difference in potential between these electrodes would produce lead I which is predominantly negative because the polarity of lead I had been reversed by the misapplication of the lead wires. The standard leads are corrected by viewing lead I as its mirror image and interchanging leads II and III. The unipolar limb leads are corrected by relabeling the lead marked aV<sub>R</sub> with the correct designation aV<sub>L</sub> and relabeling the lead marked aV<sub>L</sub> with the correct designation aV<sub>R</sub>. The leads recorded correctly are shown below. The notching of the S waves in lead V<sub>1</sub> is suggestive of an incomplete right bundle branch block; however, this is not borne out by the right chest leads as R R prime waves are not present in these leads (see below). Myocardial injury and ischemia are suggested by the slight negative S-T segments and flat T waves in the corrected lead I and in leads V<sub>3</sub> through V<sub>6</sub>. A short period of sinus arrest followed by a sinus beat is seen in lead aV<sub>L</sub>.

## Case Summary

Arteriosclerotic coronary disease with myocardial ischemia and angina pectoris. Teleoroentgenogram shows that the heart is rounded at the apex and is enlarged in its transverse diameter. There is enlargement of the great vessel shadow.



Leads properly recorded



## Answers

B C and E

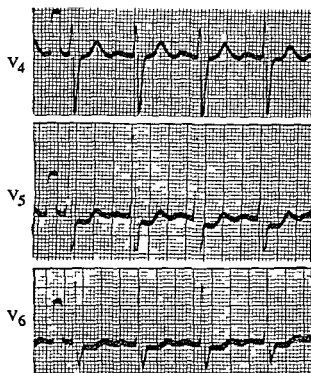
### Explanation

An incomplete left bundle branch block is present because the QRS complexes are wide and predominantly positive in lead I with small Q waves in leads I and  $V_6$ . The early electric forces of ventricular depolarization are from left to right which makes a complete left bundle branch block unlikely. Q waves are more evident in lead  $V_6$  when the electrode is elevated 1 cm (see below).

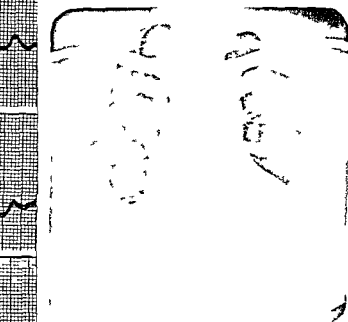
A partial AV block without dropped beats is present because of the prolongation of the P R intervals. Ventricular ectopic beats are present also. The negative J points and sagging of the S T segments in lead  $V_5$  and other leads appear to be due to subendocardial injury which could be either acute or chronic. It is of interest that these shifts have been present for three years (see below) and are in part secondary to the large areas under the QRS complexes. These shifts are not due to digitalis as at no time has this patient had this drug.

### Case Summary

Coronary arteriosclerosis, coronary insufficiency with myocardial ischemia. The heart is rounded at the apex and enlarged in its transverse diameter. There is calcification of the aorta.



Three years ago



Lead  $V_6$  elevated 1 cm

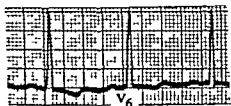
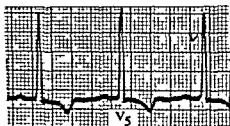
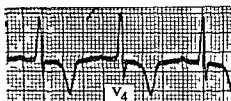
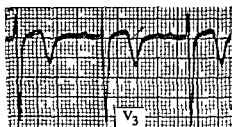
C

*Explanation*

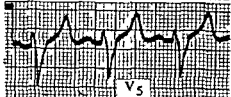
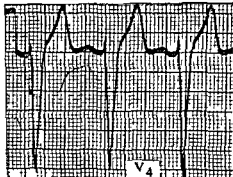
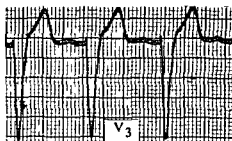
An intraventricular block is present as shown by the unusually wide QRS complexes (0.24 second). The complexes are considerably wider than those produced by an uncomplicated left bundle branch block in which the QRS width is usually in excess of 0.12 second but seldom exceeds 0.18 second. In addition, the tracing shows no demonstrable P waves, thus atrial standstill or possibly atrial fibrillation is present. A common cause for an intraventricular block is hyperkalemia; however, this was not present in this case (see summary). Low R waves in lead  $V_5$  and  $V_6$  are in keeping with a diagnosis of a posterolateral myocardial infarct which involves the interventricular septum. The diagnosis of myocardial infarction in the presence of an intraventricular block is uncertain, however, as the intraventricular block alone may sometime produce these abnormalities.

*Case Summary*

The patient had hypertensive heart disease for years. The tracing taken three years ago showed left ventricular hypertrophy and anterior myocardial ischemia (see below). The tracing taken six months ago showed a left bundle branch block in which R waves were present in leads  $V_5$  and  $V_6$  (see below). Necropsy showed an old small anterior infarct and an acute large posterior infarct extending from apex to base and involving the lateral wall of the left ventricle and the posterior wall of the interventricular septum. The non protein nitrogen was normal. The serum potassium was normal.



Three years ago



Six months ago

D

### Explanation

The tracing is in keeping with a ventricular septal defect. Electrocardiographically the tracing is suggestive of left ventricular hypertrophy because of the deep S waves in leads  $V_1$  and  $V_2$  and tall R waves in lead  $V_5$ . The fairly large U waves in lead  $V_2$ ,  $V_3$  and  $V_4$  are in keeping with cardiac hypertrophy however other states such as a slow cardiac rate and hypokalemia also produce large U waves. The diagnosis of ventricular hypertrophy can be made best when the characteristic changes in S T segments and T waves occur along with high voltage of the QRS complexes. Left ventricular hypertrophy of moderate degree which is not associated with degenerative changes of the myocardium may show high voltage of the QRS complexes without significant changes in the S T segments and T waves.

### Case Summary

Small ventricular septal defect. There is a Roger's murmur in the third intercostal space to the left of the sternum which radiates in a star like fashion in all directions and is associated with a thrill. Radiologically the heart is rounded at the apex which is suggestive of left ventricular hypertrophy. The heart is not enlarged in its transverse diameter. Right ventricular hypertrophy is suggested by the lifting of the heart from the diaphragm.



C

*Explanation*

Hyperkalemia is suggested by the tall T waves in the precordial leads. Posterior myocardial ischemia can produce similar changes; however, this often produces low T waves in lead V<sub>6</sub>. With hyperkalemia additional electrocardiographic abnormalities often develop, for example, atrial standstill, intraventricular block, and ventricular fibrillation. Sinus tachycardia is present with a rate of 125 beats per minute.

*Case Summary*

The patient had chronic glomerulonephritis. Serum potassium 7.9 mEq per liter. CO<sub>2</sub> combining power 14 mEq per liter. NPN 375 mg %. Hemoglobin 6 gm.

*Answer*

C

*Explanation*

The atria and ventricles are beating from independent pacemakers. The atrial rate is 90 beats per minute and the ventricular rate is 29 beats per minute. The atrial pacemaker is in the sinus node and the ventricular pacemaker is in the basal portion of the ventricles. An alternative theoretical explanation for the wide QRS complexes is a right complete bundle branch block with the ventricular pacemaker in the junctional tissue. This would account for the ventricular complexes which have the appearance of a right complete bundle branch block. The slow ventricular rate, however, indicates that the ventricles are driven from a pacemaker in the ventricles and not in the AV node.

*Case Summary*

Congenital complete AV heart block. No other cardiac abnormalities.





PART B

ANSWERS TO QUESTIONS ON  
ELECTROCARDIOGRAPHIC INTERPRETATION

## ARRHYTHMIAS

- 1 A Atrial depolarization is not regular so the P waves are not easily distinguishable. The R R intervals are irregular because of irregular conduction through the AV node.
- 2 A The atrial rate cannot be counted with certainty, however the atrial fibrillation waves usually occur at a rate of approximately 450 beats per minute.
- 3 A Hyperthyroidism, hypertension, coronary atherosclerosis, myocardial infarction and other disease states are often associated with atrial fibrillation or flutter. Rarely atrial fibrillation or flutter are found in patients who have no demonstrable cardiac disease.
- 4 A The ventricular rate is usually irregular especially when it is slow. When the rate is rapid the irregularity is more difficult to detect.
- 5 B
- 6 A Often the P P intervals do not vary by more than 0.01 second.
- 7 B Atrial tachycardia characteristically changes suddenly into sinus rhythm. This may be precipitated by carotid sinus pressure or deep breathing. This characteristic reaction is different from that which occurs with atrial flutter or with sinus tachycardia.
- 8 C Paroxysmal atrial tachycardia with block frequently occurs with digitalis intoxication especially if the blood potassium is low.
- 9 C Often paroxysmal atrial tachycardia occurs in apparently normal individuals without evidence of cardiac disease; however it may occur as a result of digitalis or a myocardial infarct or it may be associated with other disease states.
- 10 A Paroxysmal tachycardia includes intermittent periods of rapid heart beat when the pacemaker is in the sinus node, atrium, AV node or ventricle. Some authors might not include sinus rhythms in this group.
- 11 B Electric alternation frequently is independent of pulsus alternans.
- 12 A Frequently the AV node takes over the function of pacemaker when the sinus node is depressed.
- 13 B With AV nodal rhythm with retrograde conduction the atrial depolarization process moves away from the AV node through the atria which results in a negative field of force at the left foot electrode.
- 14 B When the node (as in nodal rhythm) is referred to it is generally meant to be the atrio-ventricular node rather than the sinus node.
- 15 D Nodal rhythm with retrograde conduction is referred to as high, middle or low depending upon the position of the pacemaker in the AV node.
- 16 A With a pacemaker in the AV node and with retrograde conduction the P waves are inverted in leads II, III and aVF. Nodal tachycardia with retrograde conduction is characterized by rapid atrial conduction occurring at a rate greater than 100 beats per minute. If the rate is between 60 and 100 it is classified as nodal rhythm.
- 17 B Retrograde conduction from AV node to atria depends upon the presence or absence of disease of the AV node and other factors.

- 18 A With parasystole an irritable focus outside of the sinus node serves as pacemaker and discharges impulses at a regular rate. The parasystolic rhythm will be the dominant rhythm if its rate is more rapid than the discharge rate from the sinus node.
- 19 A Sinus arrest is caused frequently by vagal stimulation which produces temporary inhibition of the sinus node.
- 20 A When the cardiac rate varies with respiration it is referred to as phasic sinus arrhythmia. The cardiac rate may vary with the altered activity of the sinus node which is not related to respiration in which case the term phasic is not employed.
- 21 B Phasic sinus arrhythmia is a common finding in normal children and in patients with Cheyne Stokes respiration.
- 22 A This figure has been selected arbitrarily and is accepted by most cardiologists. Many normal young adults and normal well trained athletes have cardiac rates below 60 beats per minute.
- 23 A
- 24 A These figures have been selected arbitrarily but are accepted by most cardiologists.
- 25 A
- 26 C Sinus tachycardia may be due to nervousness, anxiety, hyperthyroidism, tobacco, infections, toxemia, cardiac disease and other disease states.
- 27 A Some cardiologists may not include sinus tachycardia in this group. The term supra ventricular tachycardia is employed when a more definite diagnosis regarding rhythm cannot be made.
- 28 A Ventricular fibrillation is characterized by an absolute irregularity of ventricular depolarization.
- 29 A
- 30 A To determine the multifocal origin of ventricular ectopic beats, differences in configuration of the beats should be observed in one particular lead.
- 31 B The P waves of the basic rhythm coming ordinarily from the sinus node may be buried within the ventricular premature contractions. Also retrograde conduction following the ventricular premature contractions may occur in which case P waves will be found within or after the QRS complexes.
- 32 B Ventricular premature contractions occur in health and disease.
- 33 B The ventricular ectopic beats from the base of the heart are predominantly positive because they follow in general the normal course of ventricular depolarization that is from the base to the apex. This characteristically results in positive QRS complexes in the standard leads.
- 34 C The most common causes for bigeminy are a normal sinus beat followed by an atrial premature contraction or by a ventricular or nodal premature contraction.
- 35 B Ventricular premature contractions may occur in young individuals without demonstrable cardiac disease. They are suggestive of cardiac disease if they have their onset late in life and are precipitated by exercise.
- 36 A
- 37 A Usually the cardiac rate exceeds 100 beats per minute and the pacemaker is in Retrograde conduction through the atria may or may not occur.

- 67 A The work of the left ventricle is increased because it supplies blood to the pulmonary as well as to the systemic circulation. The amount of left ventricular enlargement is related to the size of the ductus.
- 68 A Early in life the electrocardiogram may be nearly normal. Late in life the left ventricular hypertrophy pattern is common.
- 69 B Tricuspid atresia usually is associated with an interatrial septal defect. Because of this a left ventricular hypertrophy pattern is common. In infants tricuspid atresia with non-functioning right ventricle may obscure normal physiologic right ventricular predominance picture.
- 70 B Usually a right ventricular hypertrophy picture is present. The right ventricular pressure is usually increased as the right ventricle is pumping blood against the high systemic pressure of the peripheral circulation.
- 71 C Right ventricular pressure is usually increased because of the ventricular septal defect and pulmonic stenosis and because the right ventricle is pumping blood into an overriding aorta against relatively high pressure.
- 72 A The pressure in the right ventricle and the amount of right ventricular hypertrophy varies with the degree of pulmonic stenosis. If the stenosis is slight a normal electrocardiogram may be present.
- 73 A
- 74 A
- 75 B Disseminated lupus erythematosus may or may not affect the heart.
- 76 C The electric axis of P waves often is about 90 degrees in patients with pulmonary emphysema.
- 77 C Paroxysmal atrial fibrillation or atrial flutter are common with severe untreated hyperthyroidism.
- 78 B Sinus bradycardia can be improved significantly with therapeutic doses of thyroid.

## DRUGS AND ELECTROLYTES

- 79 A Digitalis accelerates the rate of ventricular repolarization.
- 80 B Digitalis commonly causes first, second or third degree AV block but does not as a rule produce an intraventricular block.
- 81 A Characteristically digitalis does not slow the cardiac rate when an increased rate is due to toxic states or infections when congestive heart failure is not present.
- 82 A Discordant J shifts are common in most leads however in certain leads especially those near the transitional zone this general rule does not hold.
- 83 B Quinidine prolongs the time of ventricular repolarization.
- 84 A
- 85 A When atrial flutter is present with a 2:1 AV block quinidine may accelerate the ventricular rate by producing a 1:1 conduction as a result of slowing the atrial rate.

- 86 B Hypocalcemia delays ventricular repolarization
- 87 A Prolongation of the Q T intervals may be shown by measuring from the beginning of the QRS complex to the peak of the T waves In this way a fairly accurate measurement can be made
- 88 A
- 89 B With hypocalcemia U waves are common which make it difficult to measure the Q T intervals in the conventional manner
- 90 B The blood potassium level does not bear a constant relationship to the electrocardiogram, however under certain circumstances it may be of value For example in diabetic patients in coma or in post operative patients after prolonged gastric suction the electrocardiogram may be of value in determining the directional changes in blood potassium In cases of this sort, it is wise initially to take an electrocardiogram and also to determine the blood potassium clinically Thereafter the electrocardiogram may serve as a means of determining relative changes in potassium

## ELECTRIC AXIS

- 91 B The average mean electric axis of the QRS complexes for infants under 6 months of age is  $+ 130$  degrees The average for children is  $+ 52$  degrees
- 92 A The amount of right axis deviation is dependent upon the relative enlargement of the right and left ventricle and is related indirectly to the relative amount of work of the right and left ventricles
- 93 A In all of these conditions the work of the left ventricle exceeds that of the right
- 94 B When the mean electric axes of QRS and T are in opposite directions cardiac disease is usually present

## HYPERTROPHY

### (ATRIAL AND VENTRICULAR)

- 95 B The precordial leads are of value also in diagnosing atrial enlargement or hypertrophy
- 96 B Atrial enlargement should be determined from the precordial as well as from the standard limb leads
- 97 A
- 98 A The distribution of the electric forces over the surface of the body depends upon the relative preponderance of ventricular hypertrophy as well as on the position of the heart in the chest
- 99 A Although 27 mm is the upper limit of normal for R waves in lead  $V_4$  as shown in the appendix it should be remembered that the normal values are based on a relatively small series of cases
- 100 A The upper limit of normal for the time of onset of the intrinsicoid deflection in lead  $V_4$  is 0.05 seconds for adults

- 67 A The work of the left ventricle is increased because it supplies blood to the pulmonary as well as to the systemic circulation The amount of left ventricular enlargement is related to the size of the ductus
- 68 A Early in life the electrocardiogram may be nearly normal Late in life the left ventricular hypertrophy pattern is common
- 69 B Tricuspid atresia usually is associated with an interatrial septal defect Because of this a left ventricular hypertrophy pattern is common In infants tricuspid atresia with non functioning right ventricle may obscure normal physiologic right ventricular predominance picture
- 70 B Usually a right ventricular hypertrophy picture is present The right ventricular pressure is usually increased as the right ventricle is pumping blood against the high systemic pressure of the peripheral circulation
- 71 C Right ventricular pressure is usually increased because of the ventricular septal defect and pulmonic stenosis and because the right ventricle is pumping blood into an overriding aorta against relatively high pressure
- 72 A The pressure in the right ventricle and the amount of right ventricular hypertrophy varies with the degree of pulmonic stenosis If the stenosis is slight, a normal electrocardiogram may be present
- 73 A
- 74 A
- 75 B Disseminated lupus erythematosus may or may not affect the heart
- 76 C The electric axis of P waves often is about 90 degrees in patients with pulmonary emphysema
- 77 C Paroxysmal atrial fibrillation or atrial flutter are common with severe untreated hyperthyroidism
- 78 B Sinus bradycardia can be improved significantly with therapeutic doses of thyroid

## DRUGS AND ELECTROLYTES

- 79 A Digitalis accelerates the rate of ventricular repolarization
- 80 B Digitalis commonly causes first second or third degree AV block but does not as a rule produce an intraventricular block
- 81 A Characteristically digitalis does not slow the cardiac rate when an increased rate is due to toxic states or infections when congestive heart failure is not present
- 82 A Discordant J shifts are common in most leads however in certain leads especially those near the transitional zone this general rule does not hold
- 83 B Quinidine prolongs the time of ventricular repolarization
- 84 A
- 85 A When atrial flutter is present with a 2:1 AV block quinidine may accelerate the ventricular rate by producing a 1:1 conduction as a result of slowing the atrial rate

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- 01 B Normally in adults the R waves should not be taller than 7.0 mm in lead  $V_1$  or 16.0 mm in lead  $V_2$
- 02 B Left ventricular hypertrophy in a horizontal heart is associated with mean electric axis to the left. Left ventricular hypertrophy may exist in a vertical heart in which case the electric axis may be within normal limits but can be displaced to the right
- 03 A
- 04 A With right ventricular hypertrophy the transitional zone as seen in the 6 precordial leads often occurs in lead  $V_1$  or between  $V_1$  and  $V_2$ . This is shifted to the right of the usual position which is in lead  $V_2$  or  $V_3$
- 05 A If in the presence of a right bundle branch block the R prime wave exceeds 17.0 mm, it is likely that right ventricular hypertrophy is present as well

## INTRINSICOID DEFLECTION

- 06 C Delayed onset of the intrinsicoid deflections in lead  $V_5$  occurs when ventricular depolarization occurs in an abnormal fashion for example with a left bundle branch block or when the ventricular activation wave is delayed as with an intraventricular block or when the thickness of the left ventricle is increased which prolongs the time for the passage of the activation wave
- 07 B In order to obtain the most useful information it is best to place the electrodes as close to the heart as possible
- 08 B The upper limit of normal for the time of onset of the intrinsicoid deflection in  $V_1$  is 0.03 and in  $V_6$  is 0.05 second
- 09 A
- 10 A

## LEADS

- 11 A
- 12 B
- 13 C
- 14 A
- 15 B
- 16 B
- 17 A
- 18 A
- 19 B
- 20 C

- 121 B
- 122 B Normally the apex of the heart is downward to the left and toward the front of the body Lead  $V_6$  faces the posterolateral aspect of the left ventricle
- 123 A
- 124 A With atrial fibrillation an irregular base line is seen often between QRS complexes With atrial tachycardia the P waves are prominent if the electrode is properly placed and the P P intervals are nearly regular Carotid sinus pressure and deep breathing assist in differentiating between these two conditions
- 125 B The electrode is below the sinus node The atrial depolarization process moves toward the electrode which results in a positive P wave
- 126 A
- 127 A
- 128 A
- 129 C
- 130 C The  $aV_F$  electrode is common to standard leads II and III
- 131 B The  $aV_L$  electrode is common to leads I and III

## MYOCARDIAL INFARCTION

- 132 B
- 133 A
- 134 A The process of infarction is considered chronic when significant changes in the electrocardiogram no longer occur
- 135 A QS waves may occur in leads  $V_3$  and  $V_4$  with an anterior infarct and also with a left bundle branch block
- 136 A A strictly anterior myocardial infarct results in forces which travel perpendicular to the frontal plane of the body which produce very little change in the limb leads Significant changes occur usually in precordial leads
- 137 A
- 138 A When the major deflection of the QRS complex is negative the J point and ST segments are positive
- 139 B A small subendocardial infarct characteristically does not alter the height of the R waves in the precordial leads which overlie the infarct providing the epicardium is intact
- 140 C Lead  $V_3$  often overlies the anteroseptal portion of the heart if the heart is not unduly enlarged or rotated and if the electrodes have been properly placed on the chest wall
- 141 B With a small intramural infarct the electric forces produced by the infarct are equal and opposite and have a balancing effect which does not alter the QRS complexes significantly

- 142 B A transmural infarct involving the anterior portion of the left ventricle destroys the electric forces ordinarily produced by this portion of the myocardium. An electrode placed over this area records the electric forces present within the cavity of the left ventricle.
- 143 A A lateral infarct destroys electric forces on the lateral surface of the left ventricle which leaves an overbalance of forces on the right resulting in tall R waves in lead  $V_1$ .
- 144 A With a lateral myocardial infarct electric forces are moving toward or away from the  $V_5$  and  $aV_L$  electrodes; thus these leads reveal the presence of the infarct.
- 145 A Typically the Q waves are large and there are elevated S T segments and inverted T waves in lead  $aV_F$ .
- 146 A A large subacute posterior myocardial infarct is characterized by relatively deep Q waves, isoelectric S T segments and inverted T waves which change in amplitude in serial electrocardiograms. The chronic stage is reached when the amplitude of the T waves becomes constant.
- 147 C With a chronic posterior infarct Q waves are often present in lead  $aV_F$ . S T segments are nearly isoelectric and T waves do not change in amplitude in serial tracings.
- 148 B
- 149 A A negative field of force with respect to the central terminal produces a negative deflection (Q wave) in lead  $aV_F$ .
- 150 A
- 151 B An old infarct high on the posterior wall of the left ventricle may not be detected by the usual twelve lead electrocardiogram.
- 152 A Many subendocardial infarcts produce negative S T segment shifts in the precordial leads without altering the QRS complexes.
- 153 B

## MYOCARDIAL INJURY AND ISCHEMIA

- 154 A Ischemia of the subendocardium results in injury shifts which often manifest themselves by negative S T segment shifts in lead  $V_4$ .
- 155 A Coronary arteries which have narrow lumens may be able to supply adequate blood to the myocardium of the patient at rest; however, this supply may be inadequate when the patient exercises.
- 156 A The S T segment shifts often last from one to five minutes.
- 157 A
- 158 A

## PERICARDITIS

- 159 A When pericarditis is acute, severe and diffuse the S T segments are usually elevated in leads I, II and  $V_4$ .
- 160 B When the subepicardium is involved the electrocardiogram is altered. If the pericardium is involved without involvement of the subepicardium no electrocardiographic changes are produced.
- 161 A
- 162 A Tuberculosis involving the pericardium sometimes produces constrictive pericarditis which is characterized by low voltage of the QRS complexes.

# POSITIONS OF THE HEART

## (ELECTROCARDIOGRAPHIC)

- 163 B The correlation between the electrocardiographic and the anatomic position of the heart is better in health than it is in disease
- 164 B When the apex of the heart moves up (the heart becoming more horizontal) counterclockwise rotation usually occurs
- 165 A This may be associated with strong counterclockwise rotation of the heart about the long axis as viewed from the apex
- 166 A This may be associated with slight counterclockwise rotation of the heart about the long axis as viewed from the apex
- 167 A Characteristically there is no significant rotation of the heart about the long axis as viewed from the apex
- 168 A This may be associated with slight clockwise rotation of the heart about the long axis as viewed from the apex
- 169 A This may be associated with strong clockwise rotation of the heart about the long axis as viewed from the apex
- 170 A
- 171 A The indeterminate position is diagnosed when the tracing does not fit into the following categories horizontal semihorizontal intermediate semivertical and vertical

## PULMONARY INFARCTION

- 172 B S waves appear in lead I with Q waves elevated S T segments and inverted T waves in lead III Lead aV<sub>F</sub> does not necessarily resemble lead III
- 173 B Small pulmonary infarcts usually do not produce significant changes in the electrocardiogram Subacute pulmonary infarcts usually show less change than acute ones

## THEORY

- 174 A In many normal individuals the mean electric axis of the QRS complexes points away from the right arm and toward the left iliac crest In such cases the QRS complexes are small in lead III and are positive and of equal amplitude in leads I and II
- 175 A Forces moving parallel with the frontal plane of the body have little effect on lead V<sub>B</sub>
- 176 C
- 177 B
- 178 C
- 179 A Other transitional zones may be found on the body besides that which appears in the standard 6 precordial leads

- 180 A Ventricular depolarization begins from the Purkinje system which is at the endocardium
- 181 A When the depolarization process moves perpendicular to the lead I line a small or no deflection is written in that lead. If the process is moving toward the foot at a 90 degree angle with a line connecting the lead I electrode the QRS complexes will be large, positive and of equal amplitudes in leads II and III
- 182 A
- 183 C
- 184 A An intraventricular block can be local or diffuse
- 185 A This is true in general but does not hold necessarily for all of the different tissues of the body
- 186 B
- 187 A
- 188 A
- 189 A The ventricular gradient is a measurement of the electric activity (depolarization and repolarization) of the ventricles
- 190 A Electrocardiographically this would be shown as shifts of the S T segments
- 191 B
- 192 A
- 193 A A good example is a bath of saline
- 194 A
- 195 A
- 196 C
- 197 B
- 198 B

## WAVES, INTERVALS, SEGMENTS AND J POINT

- 199 C
- 200 B Notching of P waves occur in a large percentage of normal individuals
- 201 C
- 202 C See appendix
- 203 A Atrial enlargement may be simulated by rotation of the heart and should be diagnosed with caution from the standard leads. The precordial leads are of value in differentiating right from left atrial enlargement

- 204 C See appendix
- 205 B
- 206 B Prolonged P R intervals can be due to a variety of causes
- 207 B The P R interval measures the time from the beginning of atrial depolarization (P waves) to the beginning of ventricular depolarization (QRS complexes)
- 208 A Erroneous measurements may result when narrow waves or QRS complexes are used because parts of the depolarization processes may be isoelectric
- 209 A In normal subjects the Q waves are of short duration
- 210 A It is desirable to consider the amplitude of the QRS complexes in lead aV<sub>F</sub> when making a diagnosis of posterior myocardial infarction. Some normal individuals have QRS complexes of low amplitude (less than 5.0 mm) in this lead and in these subjects the Q waves may be deep with respect to the height of the R waves; however in such a case this finding is not necessarily indicative of disease.
- 211 B Relatively deep Q waves (6.0 mm or less) in lead III occur normally with obesity, left ventricular hypertrophy as well as with posterior infarction.
- 212 A Septal Q waves in lead I are small.
- 213 B Potential variations from the endocardial surface of the right and left ventricles have a strong influence on the configuration of lead aV<sub>R</sub>.
- 214 A
- 215 A Often the widest QRS complexes are found in lead V<sub>2</sub>.
- 216 A
- 217 B
- 218 A This is because the ventricular depolarization process in many normal individuals moves perpendicular to a line connecting the lead III electrode.
- 219 A Only a few normal individuals have QRS complexes wider than 0.10 second.
- 220 B Low voltage of the QRS complexes is diagnosed usually when the highest QRS amplitudes of the six limb leads is less than 5.0 mm. This occurs in the standard and in the unipolar limb leads when the ventricular depolarization process is traveling perpendicular to the frontal plane of the body. It occurs also in certain disease states, for example hypothyroidism.
- 221 A Wide QRS complexes occur with a left bundle branch block, severe hyperkalemia and from quinidine intoxication.
- 222 C The relationship is shown by the Bazett formula which is  $QT_c = \frac{Q-T}{\sqrt{R-R}}$
- 223 B Hyperparathyroidism produces hypercalcemia which shortens Q-T intervals.
- 224 B Digitalis accelerates the rate of ventricular repolarization.
- 225 A See appendix.

- 226 B Long Q T intervals may be due to quinidine hypocalcemia and other states in which organic myocardial diseases may not be present Myocardial injury and ischemia also prolong Q T intervals
- 227 B In addition to correcting the Q T interval for heart rate ( $Q T_c$ ) it is often necessary to correct for abnormal prolongation of the QRS interval i.e. in the presence of a bundle branch block
- 228 C The normal values are shown in the appendix Prolonged athletic training produces S T segment shifts which are greater than those listed in the tables of normal values
- 229 A When the segment shifts persist for as long as 24 hours necrosis of the myocardium usually results S T segment shifts lasting for five to ten minutes are associated with acute myocardial injury and ischemia however necrosis and death of muscle may not result
- 230 A
- 231 D Some normal subjects especially athletes have elevation of the R T junction which exceeds the limits of normal shown in the appendix
- 232 A
- 233 B
- 234 B Pericarditis and other disease states in addition to myocardial ischemia can produce abnormalities of the T waves
- 235 A Inverted T waves in lead I in the presence of positive QRS complexes are almost always a sign of cardiac disease providing the ECG has been taken and mounted properly Myocardial ischemia left ventricular hypertrophy and pericarditis are causes
- 236 B Secondary T wave changes are associated with increased areas under the QRS complexes
- 237 A See appendix

